

AMERICAN JOURNAL OF OPHTHALMOLOGY

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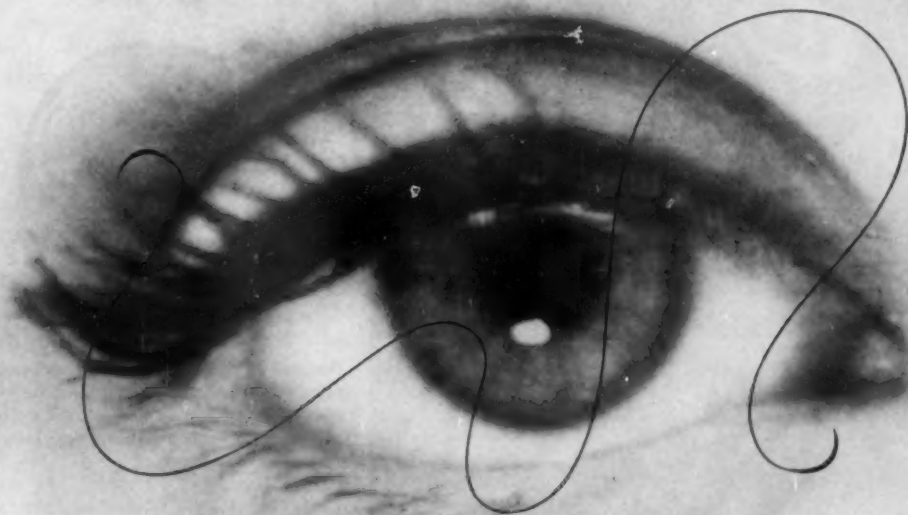
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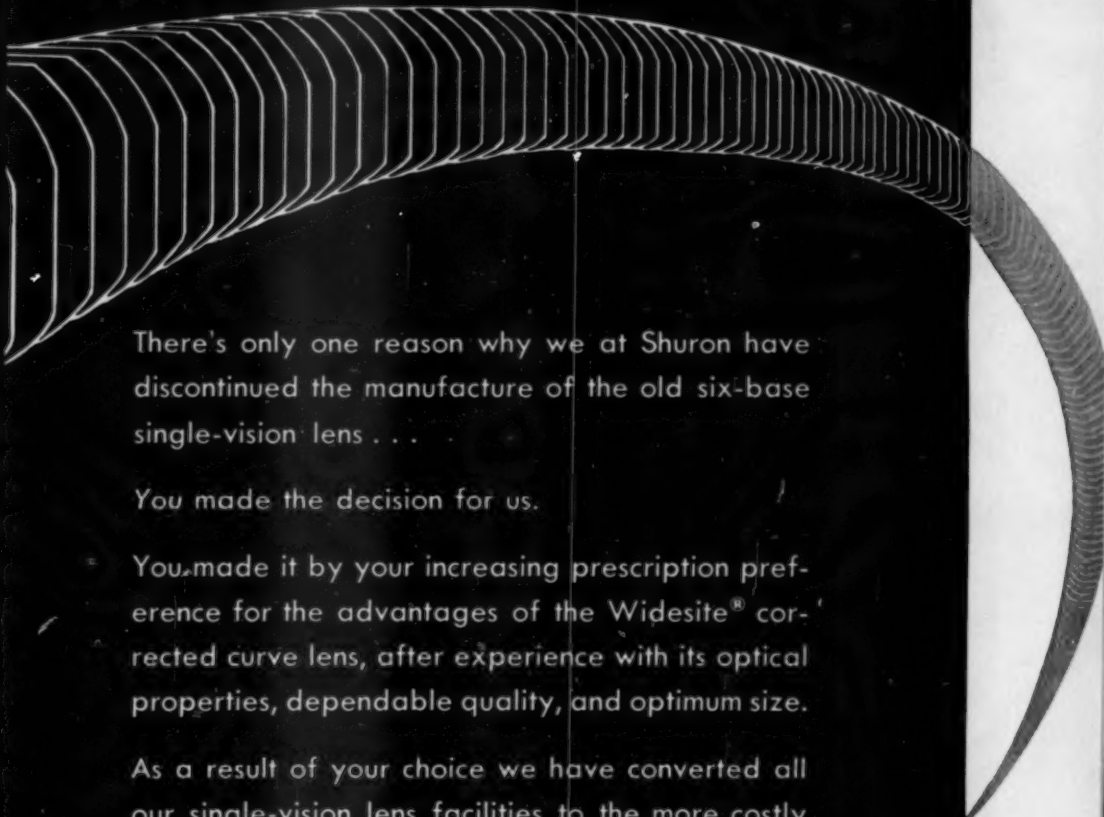


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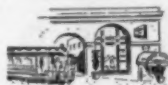
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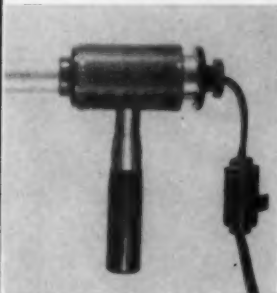
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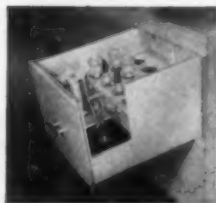
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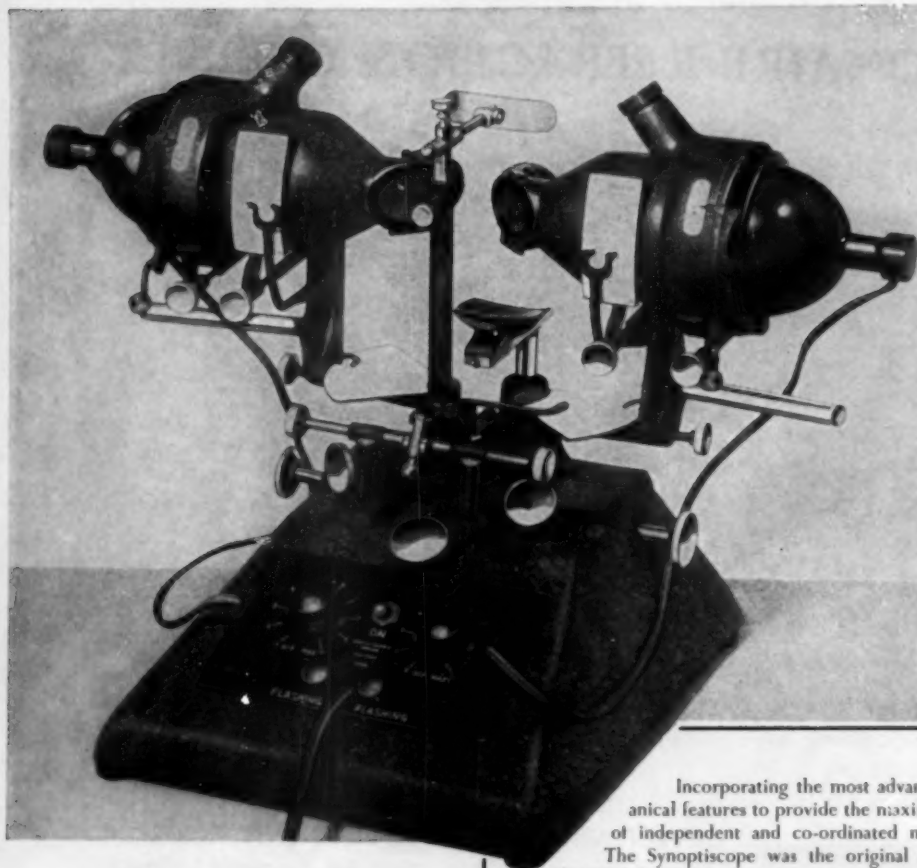
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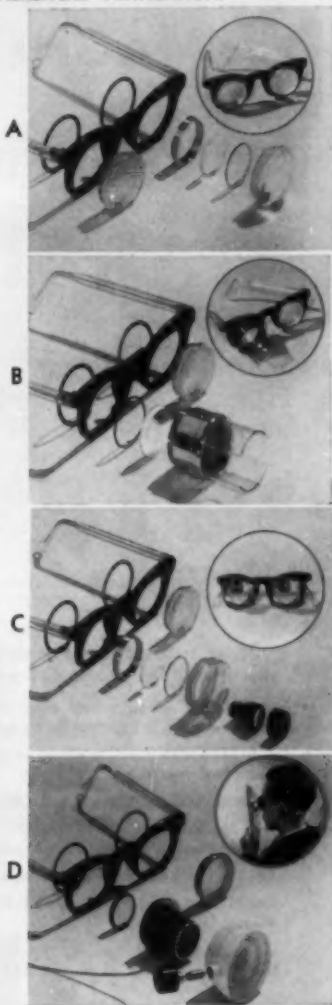
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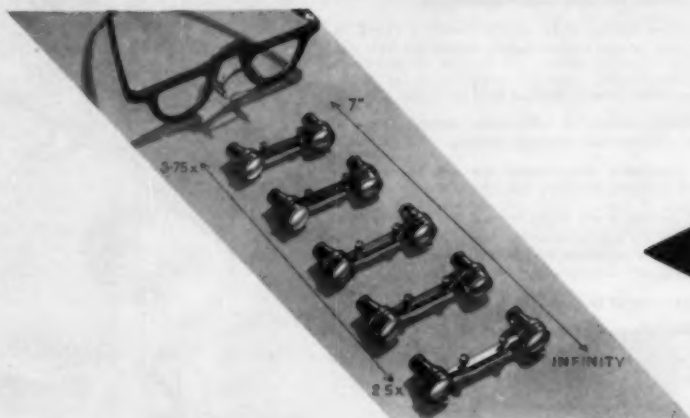


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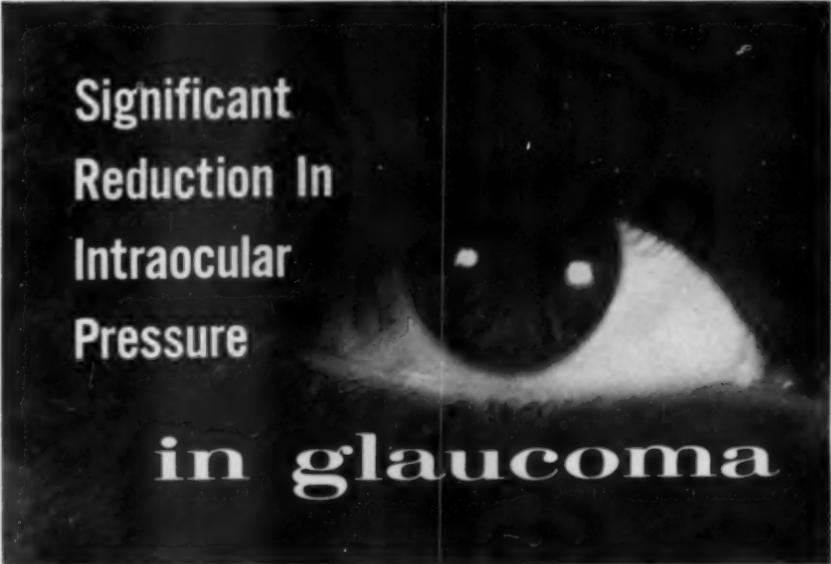
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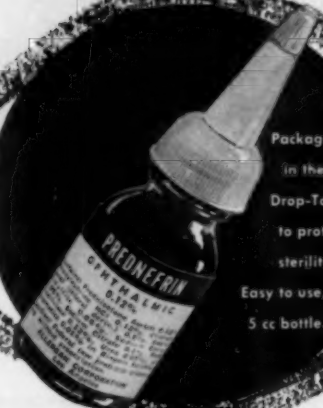
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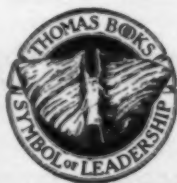
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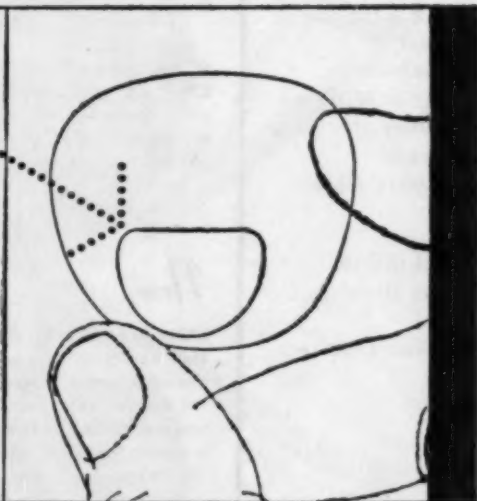
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(Figure 2)
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1. *Am. J. Ophth.* 42:771, 1956.
2. *Am. J. Digest. Dis.* 22:5, 1955.
3. *Med. Times* 84:741, 1956.

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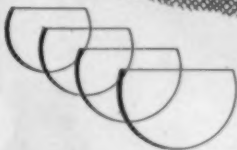
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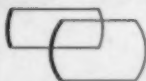
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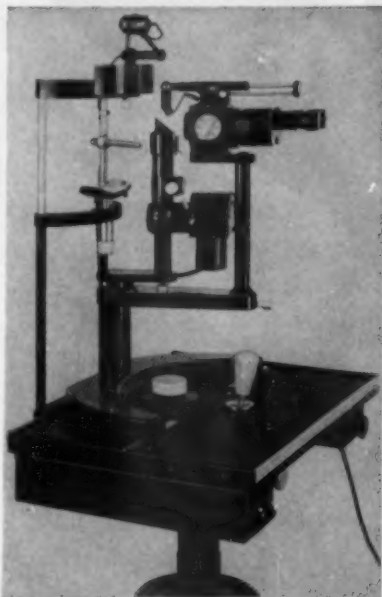
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1. Ragershek, R. H. and McIntire, W. C.: *Am. J. Ophth.* 40:34 (July) 1955. • 2. Ehrlich, L. H.: *N. Y. State J. Med.* 53:3019 (Dec. 15) 1953. • 3. Gattas, R. C.: *A.M.A. Arch. Ophth.* 51:467 (April) 1954. • 4. Council on Pharmacy and Chemistry: *J.A.M.A.* 158:1523 (Aug. 27) 1955. • 5. Stolzer, I. H.: *Am. J. Ophth.* 36:110 (Jan.) 1953.



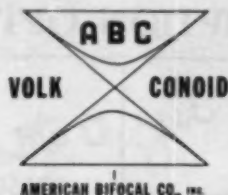
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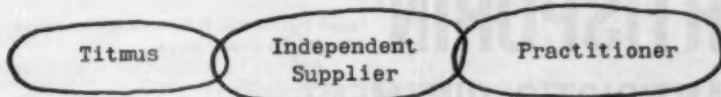
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SERIES 3 • VOLUME 44 • NUMBER 3 • SEPTEMBER, 1957

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ABSTRACTS

Anatomy, embryology, and comparative ophthalmology; General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Optic nerve and chiasm; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus; Tumors; Injuries	431
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* Abrahamson, I. A., Jr.: Acute emergencies of the eye.
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VOLUME 44

SEPTEMBER, 1957

NUMBER 3

MANAGEMENT OF INTRAOCULAR MALIGNANCY*

THE GIFFORD MEMORIAL LECTURE

EDWIN B. DUNPHY, M.D.

Boston, Massachusetts

I deeply appreciate the honor of being asked to give the Gifford Memorial Lecture. It was my good fortune to know Sandy Gifford fairly well and, like all his friends, I admired him intensely. His contributions to ophthalmology were many. Aside from his two textbooks, his bibliography contains 160 papers on a variety of ophthalmic subjects. In looking over the titles of these, I was struck by the fact that he had never written anything on ocular malignancy, so I decided to gather together some of my thoughts and experiences on this subject and present them to you this afternoon.

I have chosen for the title of this lecture "The management of intraocular malignancy" because this subject represents, I think, a great challenge to the ophthalmologist's judgment and skill. Not only are the decisions made extremely important, and often irrevocable, but, if they are correct, it is sometimes possible to save not only the patient's life but perhaps his vision as well. The same applies, of course, to extraocular malignancy but here biopsy and surgery are more often feasible and we are on much surer ground.

I shall limit the scope of this talk to intraocular neoplasms since those of the ocular adnexa, the optic nerve, and the orbit would occupy time for several more lectures. Also, I shall make no attempt to discuss the diagnosis; let us assume this has been made to the

best of our ability by the various methods available; that is, clinical appearance, biopsy, transillumination, radioactive-phosphorus test, ultrasonic waves, and so forth. We have come to the conclusion that we are dealing with a case of intraocular malignancy and the problem now is what to do about it. Shall we advise immediate enucleation or shall we consider some other methods of attack?

Our actions will depend, of course, upon various factors: such as the feasibility of removal of the tumor without sacrificing the eye, the condition of the opposite eye, the age and physical condition of the patient, the sensitivity of the tumor to irradiation or chemotherapy, the accessibility of tumor to biopsy, or to treatment with diathermy.

There are, as we all know, many different types of intraocular tumors, but, for practical purposes, the malignant ones fall into three main groups from a clinical diagnostic standpoint:

1. Retinoblastomas
2. Malignant melanomas of uveal tract
3. Metastatic tumors

I would like to discuss each group in turn.

RETINOBLASTOMA

This congenital malignant tumor of early childhood is bilateral in at least 25 percent of the cases. This fact must never be forgotten because the early discovery of the tumor in the second eye may make possible therapeutic measures designed to save life and sight. Therefore, every case of retinoblastoma must have a thorough search made of the fundus of the apparently normal eye

* From the Massachusetts Eye and Ear Infirmary and the Department of Ophthalmology, Harvard Medical School. Read at a meeting of the Chicago Ophthalmological Society, February 15, 1957.

under general anesthesia with a widely dilated pupil. To make such a fundamental statement to an audience such as this may seem quite unnecessary but what I want to emphasize is that one examination is not sufficient.

Although nothing may be found at the time, it is perfectly possible that something may show up later. Repeated examinations, therefore, should be made, under ether if necessary, every two months for the following year, and at somewhat less frequent intervals for at least another two years. It is said that 85 percent of retinoblastomas become manifest within three years, although there are a number of cases on record in which the disease has occurred much later. Another point that needs emphasizing is the multiple origins of this tumor. It is not unusual to find two or three separate tumors in the fundus. Also, vitreous and iris seeding is not uncommon and should be searched for.

It is well known that a survivor of this disease often transmits it to his offspring. Therefore, a baby of a known survivor should be routinely examined at birth, and at frequent intervals thereafter, in order to detect the condition as soon as it makes its appearance, rather than wait until the tumor has become so large that only enucleation is possible.

UNILATERAL TUMOR

It is an unfortunate fact that, when the parents first bring the child to the ophthalmologist because they have noticed the yellow-white reflex in the pupil, the tumor is so well advanced that nothing remains except enucleation. This operation should be done with certain facts in mind:

1. It is important to obtain at least 10 mm. of optic nerve.
2. It is important to inspect the muscle cone and the enucleated globe for any gross evidence of extraocular extension at the time of surgery.
3. The optic nerve should be biopsied immediately for evidence of invasion. Reese¹

has called attention to the fact that extension up the nerve itself never goes beyond 10 mm. At this point, the tumor cells gain access to the subarachnoid space and intracranial involvement probably occurs in this fashion, rather than by continuous extension via the nerve to the chiasm.

In those cases where remaining tumor is known to be present beyond the cut end of the optic nerve heroic measures are usually taken, even though fatal termination is the rule. At the Massachusetts Eye and Ear Infirmary, only one patient with involvement beyond the cut end of the nerve has survived.² She is now living and well five years after enucleation and the remarkable thing is that no special treatment was given to the orbit! No other patient with similar nerve involvement is living in spite of exenteration, x-radiation, implantation of cobalt needles in the nerve stump, or craniotomy with removal of the intracranial portion of the nerve.

Reese³ has had somewhat better luck. He advocates the implantation of radon seeds in the nerve stump.

Three gold seeds, each one being one mc. in strength, are inserted in tandem fashion. Five days later irradiation of the orbit is given three times weekly for a total of 6,000 r, in spite of the risk of subsequent contracture of lids and conjunctiva and possible obliteration of the socket. This method was used by Reese in 21 patients, of whom five were reported living and well one to 17 years after such treatment.

In view of Reese's experience, it would seem that in a case of residual optic nerve tumor, a small chance does exist of saving the patient's life by using radioisotopes locally in conjunction with X-radiation. I believe that we should give the patient the benefit of this procedure and it is quite possible that improvement in technique of properly placing these substances in the nerve may result in a greater percentage of success in the future, unless the tumor has gained access to the subarachnoid space.

Since we cannot be sure this has happened, it is better to assume it has not and act accordingly. I certainly do not favor the cranial operation to remove the nerve. To my knowledge, it has never been successful.

If inspection of the enucleated globe shows gross extraocular extension into the orbital tissues, the prognosis is extremely poor.

Some recommend exenteration of the orbit in such cases, but I have never seen a successful result and do not favor this mutilating operation except for the relief of pain or for cosmetic reasons. Reese⁴ reports he has treated 25 patients with recurrent disease of the orbit by exenteration and X-ray therapy and all died. We have had one with definite extrascleral extension, but without nerve involvement, who was given a course of X-radiation for a total of 3,900 r and survived! He is living and well today, two years after therapy with no sign of recurrence or distal metastases.

It must be borne in mind that distal metastases in bones and viscera may occur just about as frequently as intracranial extensions. The treatment of these metastases by surgery or irradiation, although hopeless, is justified only because if left in situ they may constitute an unbearable situation.

BILATERAL CASES

If there is extensive intraocular growth in both eyes when the patient is first seen, bilateral enucleation, horrible though it is, must be considered and the parents allowed to make the decision. Fortunately, in most instances of bilaterality, one eye is much farther advanced than the other. Therefore, after enucleation of the more advanced eye, there are several steps that should be considered to save the remaining eye. The three main methods of treatment are:

1. Irradiation
2. Chemotherapy
3. Diathermy

Retinoblastoma, being a fairly radiosensitive tumor, is amenable to irradiation therapy but the dosage must be carefully controlled.

Unfortunately, in the past, the dosage of X-rays used to destroy the tumor was either too small to have much effect or so large that it played havoc with the extremely radio-sensitive lens, ciliary body, and cornea. About 20 years ago, Martin and Reese⁵ reported their method of fractional X-radiation, using special cones to direct the beam to the posterior pole of the eye, thereby sparing these more susceptible tissues in the anterior segment. Treatments were given three times weekly, using alternate portals for a total dosage of 8,000 r \times 2.

In Reese's follow-up of the patients treated by this method about 50 percent survived five or more years and half of these retained some vision.⁶ There were, however, some untoward late results of this rather heavy irradiation, such as vitreous hemorrhage, bony changes, and contraction of the orbit. Fortunately, chemotherapy became available and its synergistic action with X rays has opened up new hopes for curing these patients.

In 1953, Kupfer⁷ first reported the combination of X-ray therapy and intravenous nitrogen mustard in the treatment of retinoblastoma. The nitrogen mustard was used to depress the vitality of the cancer cell, rendering it more sensitive to smaller dosages of X-radiation.

Since then, Reese and his associates⁸ have treated 57 patients by a combination of triethylenemelamine, an analogue of nitrogen mustard, and X-ray therapy, using less than half the irradiation originally given when X-radiation alone was used, and, thus, avoiding most of the undesirable late effects. Triethylenemelamine has the advantage of having a specific effect on retinal cells but must be used with X rays to be effective. With this combination, they report 70 percent of their cases arrested, compared to 50 percent arrested with X rays alone. Best results are obtained in small- and medium-sized tumors.

Triethylenemelamine is usually given intramuscularly in a dose of 0.1 mg. per kg. of body weight. Twenty-four hours later a

course of X-ray treatment is begun. At the conclusion of this, triethylenemelamine is administered again followed by another course of X-ray therapy. A third dose of triethylenemelamine is then given. The white count and platelet count must be watched carefully and achromycin is started if the white blood count gets down to 2,000. For far-advanced tumors, triethylenemelamine is injected directly into the internal carotid artery.

Aside from X-ray therapy, other methods of irradiation have been used from time to time and should be mentioned. In 1931, Moore, Stallard, and Milner⁹ of England first reported the successful use of the implantation of radon seeds in the sclera over the tumor. This marked a milestone in the use of radioactive materials to attack retinoblastomas. This method was not used extensively but has recently become popular again in Australia through the work of Joyce and Scott.¹⁰

Other methods consisted of inserting radium needles or radioactive tantalum wire into the orbit, but the results were not good because of inability to control the dosage. More recently, Stallard¹¹ has employed cobalt discs with apparently excellent success. These radioactive discs, molded to fit the curvature of the sclera, have a platinum casing, which can be sewn onto the sclera directly over the tumor. Various sizes are provided for different tumor growths.

Cobalt⁶⁰ is an unstable atom, which disintegrates to nickel, giving off both gamma rays and beta particles. The platinum case stops the beta, allowing only the gamma to get through. The toxicity of both cobalt and nickel to tissues is prevented by the hermetically sealed case.

Left in place one week, the dosage is calculated to give 3,500 r to the apex of the tumor and 19,000 r to the base. With this rather high dosage, the same complications are seen as with heavy X-radiation but, according to Stallard, they are not as frequent or as severe. The principle involved is to hit the target accurately and hard with as little

scattering of radiation as possible to other parts of the eye.

In Stallard's series of 23 patients treated in this manner 21 have survived and in 12 of these the vision is fairly good. This is definitely better than his results with radon seeds. Stallard feels that, when less than one third of the retina is involved, there is reasonable hope that the growth may be destroyed by these discs. One wonders if, in the case of multiple tumors, this method would be applicable.

We now come to the treatment with diathermy, a method which has not achieved much popularity in this country and, yet, one which deserves consideration. Weve¹² of Utrecht, in 1932, was the first to employ it to attack certain retinoblastomas in the remaining eye after the first eye had been enucleated. He treated 16 patients, some combined with small doses of X rays. The tumor was destroyed in seven but only two retained reasonable vision.

In this country, the first report of the use of diathermy in retinoblastoma appeared in 1951, when Perera¹³ described a bilateral case in a seven-year-old boy. One eye had been enucleated with pathologic confirmation of the diagnosis. The tumor of the other eye was situated in the periphery and showed increasing growth under observation. The anterior location of this tumor made X-ray treatment undesirable. The tumor was, therefore, thoroughly cooked by perforating diathermy, each application lasting three seconds, using 40 ma. of current. In spite of this rather severe treatment, convalescence was uneventful and now, six years later, Dr. Perera informs me the boy is alive and well and has 20/15 vision with a field defect corresponding to the area of retinal scarring.

CASE REPORTS

I would like to mention very briefly six cases of bilateral retinoblastoma that I have seen at the Massachusetts Eye and Ear Infirmary in which this method has been successfully employed. In two others it was unsuccessful. These cases are to be reported

in detail in a separate paper, so I will present them here only in abstract form.

The six patients who were successfully treated have been followed from one to 17 years with amazingly few opacities in the vitreous and with good visual acuity in all those old enough to be refracted. There are several others treated in the past year who, so far, seem to be doing well but the period of observation is too short to classify them as successes.

In the ones which were unsuccessfully treated, the tumor grew relentlessly in spite of thorough diathermy coagulation. In one case, the tumor was near the optic nerve and very difficult to reach, particularly in a child's orbit. The other one developed seeding in the anterior chamber.

Our method is as follows:

The sclera is bared, detaching recti muscles if necessary to reach the area of tumor. The use of indirect ophthalmoscopy greatly aids accurate localization. The sclera is indented over the tumor with a flat diathermy electrode as the ophthalmoscopist is observing the lesion. A mark is made by nonperforating diathermy. Further marks are made around the edge of the lesion under ophthalmoscopic control.

When the tumor is completely surrounded, perforating diathermy is then used in the center of the ring, employing 2.0 mm. needles and leaving them in for at least 10 seconds, using a 30 milliampere current. A number of perforations are made over the area, depending on the size of the tumor. Air bubbles may appear in the vitreous if the electrode penetrates beyond the tumor but this is of no consequence.

The immediate reaction to this procedure is swelling of the retina and the appearance of hemorrhages on the surface of the tumor. The tumor itself becomes more gray-looking during the next few days but later on begins to shrink in size. Vitreous opacities appear, due probably to sloughing off of necrotic tumor cells. The anterior segment of the eye shows practically no reaction. Gradually the hemorrhages and vitreous opacities disap-

pear, leaving a scarred atrophic area which is pigmented around its borders. It is remarkable how well the eye tolerates this treatment, which can be repeated if necessary.

No one has accumulated enough cases of retinoblastoma treated by diathermy to determine whether this is the method of choice for treating the remaining eye in bilateral cases. It has the advantage of direct attack, instead of the time-consuming combination of X-radiation and chemotherapy. It is much simpler than the attachment of cobalt discs or the implantation of radon seeds. In small flat tumors, situated in the periphery of the fundus where X-radiation is hazardous, it seems logical to try it. In tumors near the optic nerve, or in very large tumors, or in those which show seeding in the vitreous or the iris, it is not recommended.

MALIGNANT MELANOMAS

It is generally conceded that these tumors are not radiosensitive and almost all ophthalmologists recommend immediate enucleation of the eyeball when the diagnosis of malignant melanoma of the choroid or ciliary body has been made. However, there are certain confusing facts which have made some wonder whether, in certain cases, we should not consider some other form of attack.

It is well known that cells from a malignant melanoma penetrate the blood vessel walls very early. This was observed by Wintersteiner¹⁴ as far back as 1907 and has been emphasized by Fuchs,¹⁵ by Papolczy,¹⁶ and by Reese.¹⁷ Therefore, it is probable that early in the disease before enucleation the whole body is deluged with tumor cells. Why, then, do not all cases show metastasis? Are there certain defensive substances in the body which restrain the metastatic cells for a period of years and does this apparent immunity finally diminish in some cases with aging of the tissues, resulting in appearance of metastatic lesions? No one knows.

Certainly there are some small choroidal tumors which appear in distant parts even after early enucleation, whereas some larger

ones may remain in situ for years and never metastasize.

I am reminded of one patient of mine, now aged 59 years. Twenty years ago she was told by three Boston ophthalmologists that she had a malignant melanoma of the ciliary body and she must have an enucleation to save her life. One of the eminent doctors went so far as to say that if the eye were not removed within a week she would be dead within a year! She refused operation and 14 years later was first seen by me because of a blind painful eye.

The globe was stony hard, the lens cataractous, and choroidal tissues was extruding through the sclera. I performed an enucleation and pathologic examination showed a malignant melanoma of the ciliary body and root of iris with extensions through the sclera along an anterior ciliary vessel. The three ophthalmologists had been quite right in their diagnosis but the only trouble was that they were now all dead, whereas the patient was in excellent health. General examination revealed no evidence of metastatic disease. Another six years have now elapsed and she continues to have robust health, with no orbital recurrence or distal metastases.

There are a few other well-authenticated cases of similar nature, even with more malignant cell types than in my case, showing that one cannot prognosticate the life span when dealing with an individual. However, according to the statistics of von Hippel¹⁸ and Papolczy,¹⁶ the mortality figures seem to vary with the stage of the disease at which enucleation is performed. For instance, in the first stage the mortality is 33 percent; in the second stage, 45 percent; and in the third stage, 90 percent. This would seem to indicate that, in general, the earlier enucleation is done the better the prognosis, but over-all statistics show that about 48 percent of all patients with malignant melanoma of the choiroid are dead within five years after enucleation.

It is known, of course, that certain cell types are more malignant than others. Ac-

cording to Callender's¹⁹ classification, the spindle-cell A and B and the fascicular type have a mortality of about 20 percent, whereas the necrotic, mixed, and epithelioid type have a mortality of about 60 percent. Another factor which contributes to the prognosis is the argyrophil fiber content of the tumor.²⁰ However, these factors cannot be determined pre-operatively by ophthalmoscopic examination. Therefore, the safest procedure seems to be enucleation, but, in certain unusual circumstances, some other form of treatment, such as diathermy coagulation, should be considered.

Again Weve²¹ of Utrecht was the pioneer here, as he was in the therapy of retinoblastoma. According to his latest report, he has used diathermy on 17 patients with a clinical diagnosis of malignant melanoma of the choroid, of whom only two died of metastatic disease within five years. Weve states that, although this series is a small one, a 12 percent mortality after diathermy compares very favorably with the 48 percent mortality after enucleation. One may question the validity of drawing any conclusions from these figures because it has not been proved histologically that the lesions he treated with diathermy were actually malignant. Also, it seems strange that, since malignant melanomas of the skin respond so poorly to diathermy, we should expect the choroidal ones to respond so favorably.

At the Massachusetts Eye and Ear Infirmary, we have attempted diathermy coagulation in only one patient with a clinical diagnosis of malignant melanoma of the choroid:

This young lady was first seen by Dr. Virgil Casten in 1948 at the age of 25 years. The left fundus showed a greenish gray, slightly elevated lesion about the size of the disc just below and temporal to the macula. The visual acuity was 20/30-. The right eye was completely normal with 20/20 vision. After a period of observation, the consensus was that she had a malignant melanoma of the choroid and enucleation was ad-

vised. This she positively refused but she did consent to diathermy treatment, even though she was told this procedure might jeopardize the macula.

The lateral rectus and the inferior oblique were detached. The lesion was located by spot transillumination and indirect ophthalmoscopy. It was then thoroughly cooked with both surface and penetrating electrodes. A considerable reaction followed with hemorrhages over the surface of the tumor, and later the area filled in with extensive pigmentation.

The patient has been followed at frequent intervals for the past eight years and no progression of the tumor has occurred. Unfortunately, the macula was damaged in the procedure, which was not anticipated.

Again, in this case, we have no proof that the lesion was malignant. Assuming that it was, it is still possible that a metastatic focus may be lying dormant in the liver and may show up later on.

Although I sometimes wonder how many lives we actually save by enucleation, I would still advocate it in most all cases of malignant melanoma of the choroid but in certain special circumstances, such as a one-eyed individual, or the fellow eye being amblyopic, or a patient absolutely refusing enucleation, I think it is justified to employ diathermy coagulation if the tumor is accessible and not too large. One risks, of course, the extension of tumor cells into the orbit along the path of the diathermy puncture if penetrating electrodes are used and it is for this reason that Weve usually advocates only surface diathermy. Actually, I have seen this in two patients with malignant melanoma of the choroid on whom diathermy punctures had been done on the mistaken diagnosis of serous retinal detachment.

If diathermy is chosen for the treatment of these special cases, Weve recommends the following principles. Only small tumors, not too close to the optic nerve, should be attempted. Usually surface diathermy is preferred, except when the tumor protrudes

well into the vitreous. Accurate localization on the sclera must be made and the tumor treated under ophthalmoscopic control. Low intensity of current should be used and the tumor coagulated for five to 10 seconds per application. The process may be repeated if there is still a suspicious area.

If enucleation for malignant melanoma is performed, the same principles should govern the surgical technique as in retinoblastoma; that is, gentle dissection of the capsule, securing a long piece of optic nerve, especially in tumors near the optic disc, and inspection of the orbit and enucleated globe for any gross evidence of extraocular invasion. If no gross extension is found, or if a small extension is well encapsulated, an implant may be used. If frank orbital tumor is present or if it recurs in the socket after enucleation, exenteration should be done forthwith even though the prognosis in such cases is extremely poor.

The use of chemotherapy in malignant melanoma is in its infancy. Farber and his associates²² at the Children's Medical Center in Boston report its use in eight patients with metastatic disease from sources other than the eye, with temporary improvement in only two.

At the Massachusetts Eye and Ear Infirmary, we had one patient with choroidal melanoma, who showed metastatic lesions in the liver, lungs, and bones 12 years after enucleation. She was given a course of treatment with triethylenephosphoramide (TEPA) with definite improvement for 18 months but death eventually occurred.

IRIS TUMORS

There is general agreement that melanotic tumors of the iris are not usually as malignant as those of the ciliary body and choroid. Some say this is because they are discovered earlier before they have a chance to grow very large. Certainly, their accessibility makes them more amenable to excision. If the gonioscopic examination shows that the tumor does not involve the ciliary body and

that there are no implantation growths elsewhere in the angle, surgical removal is indicated but every caution should be taken to see that the lesion is entirely removed.

In performing excision of an iris tumor, it is best, I think, to use a keratome incision to one side of the growth. The limbal incision can then be enlarged by scissors, taking care not to touch the tumor. The iris is grasped to one side of the tumor, drawn out of the anterior chamber, and a radial cut made to the base. The iris is then torn free from the ciliary body and another radial cut made on the other side of the tumor. Reese advocates the use of a traction stitch in the corneal lip of the incision, so that it may be lifted up as the iris, with the tumor, is being drawn out of the anterior chamber.

It sometimes happens that pathologic examination shows that the iridectomy has not completely removed the tumor, in which case enucleation should be performed if a true malignant melanoma has been found in the biopsied specimen.

In those cases in which gonioscopy shows that the ciliary body is definitely involved or that implantation growths are present in the angle, enucleation should be done. There are some cases reported in which an attempt has been made to excise the ciliary part of the tumor also but usually with no lasting results. I do not recommend diathermy treatment of iris and ciliary body tumors.

OPTIC DISC TUMORS

True malignant melanoma of the optic disc has been reported a number of times, although most so-called cases originate from the adjacent choroid. In any event, enucleation is probably indicated since any other form of treatment is impossible and because the tumor may spread rapidly up the optic nerve. These are difficult decisions to make, especially if the visual acuity is good. In many cases, the patient comes to our aid by refusing enucleation.

METASTATIC TUMORS

Metastatic tumors occur in the eye occa-

sionally, usually from carcinoma of the breast, although many other primary sites have been reported. In the past, when confronted with such a case, the ophthalmologist's attitude has been one of hopelessness, either doing nothing or occasionally treating the involved eye by irradiation, chiefly for morale purposes.

More recently, the recognition that certain tumors (especially those of the breast and prostate) can be influenced by hormonal environment has given rise to hopes that many unfortunate individuals may have their lives prolonged and eyesight improved at least temporarily.²³ There are now on record several cases of metastatic disease of the eye definitely benefited by this method of treatment, so that I think all ophthalmologists should be familiar with the principles involved.

General surgeons have long known that androgens or sterilization could temporarily control carcinoma of the breast in many cases, whereas estrogens or castration were helpful in carcinoma of the prostate. However, some curious contradictions began to appear. For instance, stilbesterol, while helping most cases of prostatic carcinoma, occasionally helps cases of carcinoma of the breast also. This is especially true in postmenopausal patients, whereas in premenopausal patients it may light up the disease.

The reports in the ophthalmic literature are meager. Ellis and Scheie²⁴ in 1952 reported marked regression in bilateral choroidal metastases in a case of a woman with cancer of the breast following sterilization by X-radiation.

In 1954 Cogan and Kuwabara²⁵ reported a case of breast cancer with choroidal metastasis to one eye, which increased despite testosterone but regressed markedly with stilbesterol for a two-year period.

This confusing picture has been cleared up to some extent by the work of Huggins and his associates^{26, 27} of the Ben May Laboratories for Cancer Research in Chicago. They have shown that after oophorectomy there is a compensating hypertrophy of the adrenals

and increased cortical function. Thus, many women continue to excrete estrogenic substances even after surgical castration but these usually disappear following bilateral adrenalectomy. Likewise, in men castration is followed by an increase of the 17-ketosteroids in the urine and these can be eliminated by bilateral adrenalectomy. Thus, there developed the combination of bilateral oophorectomy and bilateral adrenalectomy in the hope that these operations would eliminate all sources of hormones that might possibly favorably influence the metastatic growth. Such patients, of course, require cortisone for the rest of their lives. Not all cases respond because apparently some tumors are not as hormone dependent as others.

Mr. E. F. King²⁸ of Moorfields reports the case of a woman, aged 53 years, who, one year after mastectomy for breast cancer, developed a large choroidal metastasis, along with extensive pulmonary involvement. A bilateral oophorectomy and adrenalectomy was performed and there followed a complete disappearance of the choroidal metastasis within three weeks and an almost complete disappearance of the pulmonary involvement within three months. One year later the patient was well.

King reports another patient with cancer of the breast and choroidal metastasis who has done remarkably well for the past three years, following combined surgery on ovaries and adrenals. The choroidal metastasis receded markedly.

A further step in the hormonal control of mammary and prostatic cancer was taken in 1953 by Luft and Olivecrona²⁹ of Stockholm, who performed hypophysectomy in an attempt to eradicate all sources of gonadotropic and somatotrophic hormones and the removal

of various possible growth factors. In this way the removal of the pituitary might have a greater field of usefulness than adrenalectomy. So far, the metastatic breast cancers treated in this fashion have responded fairly well and the operation is well tolerated in the hands of capable neurosurgeons. In a review of the literature on this operation, I could find no case which showed ocular involvement but it is probable that metastatic eye lesions will also be controlled by this procedure.

It was hoped that, since the pituitary has a melano-stimulating hormone, the operation might favorably influence malignant melanomas but this has not proved to be the case in four patients with this tumor occurring in other parts of the body.

The time is coming, I think, when we can take a much more hopeful outlook on metastatic carcinoma of the choroid and get our patients quickly to the general surgeon or neurosurgeon, who may be able to help them a great deal, at least on a temporary basis.

CONCLUSION

In conclusion, I have tried to collect and evaluate some of the newer concepts of attacking intraocular malignancy other than by enucleation. It is not my purpose to condemn this time-honored procedure but merely to remind you that some alternatives exist, which may be applicable in certain special cases. It is my feeling that, with the advancement of scientific knowledge, the development of chemotherapy, the use of radioisotopes, diathermy, and hormonal controls, fewer tumor-containing eyes will need to be removed in the future.

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MANAGEMENT OF THE PRIMARY GLAUCOMAS*

THE ARTHUR J. BEDELL LECTURE

JOHN M. McLEAN, M.D.

New York

It is both a pleasure and a privilege to give the Bedell Lecture at the annual Wills Meeting. I have admired Dr. Bedell's many achievements in ophthalmology for a long time and I have always had a soft spot in my heart for the Wills Eye Hospital. I believe that I have attended all but one of your annual meetings, since their inception. Absence from the country prevented me from attending that one.

Selection of a topic worthy of the names "Bedell" and "Wills" has not been an easy one. Obviously it had to be one of major ophthalmologic importance. No one else could discuss Dr. Bedell's favorite subject, fundus photography, in a manner which would do justice to his established mastery of that subject. Preferably, to do justice to both names, the topic should deal with new material and, preferably, because of Dr. Bedell's well-known spirit, it should not be a subject free from controversy.

A discussion of the management of the primary glaucomas would seem to fit these criteria. Glaucoma, the leading cause of adult blindness, is certainly an important subject for ophthalmologists. Its management has changed considerably in recent years with the introduction of newer methods of study and innovations in therapy. In fact it has changed a great deal not only during Dr. Bedell's distinguished career in ophthalmology but even during my own much shorter period of service in our chosen specialty. Finally, it is a subject in which there is still plenty of room for honest difference of opinion, productive debate, and future development.

If we look at the management of glaucoma in the early 1900's, half a century ago, we find significant new developments in the introduction of the filtering operations, iridectomy then being already a half-century old. A few simple miotics were available. They, plus iridectomy, enucleation, and a few homes for the blind, comprised almost the total armamentarium for glaucoma management at the turn of the century.

The filtering operations, both external and internal, caused a surge of surgical enthusiasm. As I look back on my own introduction to ophthalmology nearly a quarter of a century ago, I find that very little further advance had been made. I often heard the dictum, "The indication for operation in glaucoma is the establishment of the diagnosis."

Ophthalmic surgeons frequently accosted each other with the question: "What are you doing for glaucoma?" The answer was usually that surgeon's favorite operation, all too frequently applied to all cases. The literature of the day was replete with series comparing this, that, and the other operative technique in "glaucoma."

Little attention was paid to the details of the disease and few classifications were used. Sometimes distinction between "primary" and "secondary" and sometimes separation into "acute" and "chronic" were made. By and large, glaucoma was glaucoma and you treated it by your favorite operation if you could, by miotics if your patient refused operation. I like to think that we are doing much better today, although I shudder to consider what the ophthalmologists of 25 or 50 years hence may have to say about our current ideas.

First, let me discuss terminology and classification. We, of the "mechanistic school," like to think of one type of primary

*From the Department of Surgery (Ophthalmology) of the New York Hospital—Cornell Medical Center. Delivered at the Ninth Annual Clinical Conference of Wills Eye Hospital, February 9, 1957, Philadelphia, Pennsylvania.

glaucoma which occurs in eyes with shallow anterior chambers and narrow angles as the result of occlusion of the filtration angle by iris tissue. We are prone to speak loosely of "narrow-angle" or even "shallow-chamber" glaucoma. I would quarrel with these terms and insist on either "angle-block," "angle-closure," or some similar term. As I shall indicate presently, not every shallow chamber nor every narrow angle is subject to angle-closure glaucoma.

We also like to think of a type of primary glaucoma which occurs without any iris encroachment on the trabecular structures. This disease often occurs in eyes with deep anterior chambers and wide angles. We are thus sometimes led to refer to "deep-chamber" or "wide-angle" glaucoma. Again, let me act the purist and insist on "open-angle" glaucoma or some immediate synonym. I am not being purely contumacious but trying to use a term which does not, by definition, rule out the occurrence of the open-angle mechanism in an eye with a chamber which is not deep and an angle which is not wide. Although such a combination may not be common I am convinced that it does exist, that classification cannot be made on chamber depth measurements or angle width estimates alone, and that a combined disease of angle closure superimposed on pre-existing open-angle glaucoma can, rarely, but really, occur. I would, then, classify the primary glaucomas which I have studied into: open-angle (the most common type), angle-closure (somewhat less frequent in occurrence), and a combined type (rare).

Is this classification therapeutically significant? Does such refined diagnosis beyond the recognition that the patient has primary glaucoma help in his management? I feel most emphatically that it does.

The older teaching was well summed up by Elliott¹ in 1918 when he said, "All forms of glaucoma . . . are eventually brought about by a closure of the filtration angle." This general opinion persisted for a long time in spite of Henderson's statement,²

"... blocking of the angle is a constant and marked pathologic feature in acute glaucomas; while in the chronic noncongestive forms it is absent, and the angle is found open." Both von Graefe³ and de Wecker⁴ recognized amaurosis with excavation of the disc in which iridectomy was futile but tended to separate these cases from glaucoma.

To Barkan, in this country, belongs the credit for popularizing gonioscopy and demonstrating the role of angle blockage in certain of the glaucomas as well as its absence in many others. His early papers on this subject⁵⁻⁸ are classics in the field of glaucoma and, I am sure, well known to all of you.

Early in my residency training I became interested in gonioscopy as a means of studying glaucoma and of investigating some of the alterations of the angle structures produced by various types of surgery. In fact, at the same meeting, Kronfeld and Grossman⁹ and I¹⁰ reported independently on the mechanism of aphakic glaucoma after cataract extraction as disclosed by gonioscopic studies. Gonioscopic elucidation of the mechanism of the glaucomas has been gaining popular acceptance in the last two decades.

More recent dynamic studies of aqueous flow have ably supported the static evidence of angle anatomy and given us further tools for the study and rational treatment of the glaucomas. Stimulated by Friedenwald, Moses and Bruno¹¹ pointed the way to quantitative estimation of aqueous outflow in the intact eye and Grant,^{12,13} also aided by Friedenwald, gave us our present refined methods of tonography. Unfortunately, accurate tonography is not yet widely available outside the larger ophthalmic centers but reasonably good approximations by simplified methods, as outlined by Grant,¹⁴ are within everyone's reach.

Failing this, the individual practitioner can at least get a rough approximation of the outflow status in an eye by the use of Blaxter's¹⁵ bulbar pressure test and outflow factor. This last method does not require any

expensive equipment nor elaborate computation. At the present time there is some confusion in relation to absolute values of aqueous outflow just as there is some confusion about absolute interpretation of tonometric readings.

This state of affairs has been brought about by attempts to refine the estimates of intraocular pressure. The newer tables put out by the Committee on Tonometer Standardization are an attempt to reflect more accurately the true facts of absolute interpretation of tonometer readings but they have been misinterpreted in some quarters. It is important to realize that any given scale reading with any particular weight on a standard Schiötz tonometer is unchanged by the newer tables of manometric interpretation. The translation into millimeters of mercury may be different but with that difference goes a corresponding shift in our concept of the normal ranges. Any scale reading which was normal is still normal and any scale reading that was abnormal is just as abnormal, no matter what we may call it in terms of pressure equivalents. I must plead guilty, as a member of that committee, to having a part in causing this confusion.

I believe, nevertheless, that the temporary discomfort produced by restandardization is justified for it allows us to get closer to the facts in computation of aqueous hydrodynamics by tonographic methods and gives a very comforting closeness of correlation between tonographic studies, fluorescein transfer, and chemical turnover measurements. At the moment, the tonographic literature is a bit muddled for various investigators have used different standards in computing coefficients of outflow and their numerical values are not necessarily transferable without mathematical correction. I shall, therefore, in this paper studiously avoid absolute terms in dealing with pressures and outflows. Let each investigator and each clinician compare his values with his standard norms until greater unanimity is achieved and we all talk the same language. I trust

that the time when we do is not far off.

With this preamble let us look at the clinical management of the primary glaucomas and consider angle closure glaucoma first. This may be an acute situation with very high pressure, marked congestion, and all the well known signs and symptoms; a less severe but relatively acute episode; an interval phase between attacks; or a recognizable potential problem, often in a fellow eye, which has not yet become manifest. The acute attack requires emergency management with the intensive use of strong miotics topically, adequate systemic administration of carbonic anhydrase inhibitors, and sedation.

Occasionally, retrobulbar block and hypertonic intravenous infusions may help. Gonioscopy at the acute stage will show only complete angle closure without revealing any detail beyond. Tonography will show virtually no outflow. Such findings early in the attack do not necessarily indicate the status of the angle or outflow mechanism beyond that immediate moment. It must be remembered that moderate turbidity of the aqueous and an occasional inflammatory cell are compatible with acute primary glaucoma and do not arbitrarily indicate secondary glaucoma from primary inflammatory disease.

Operative intervention in this acute phase is fraught with many hazards and, since the introduction of Diamox, has rarely been necessary. As long as a reduction in tension is being obtained, it seems justifiable to continue conservative nonsurgical measures until the attack is controlled. Success in this endeavor does not represent an operation avoided. Surgery is clearly indicated in any eye in which the angle has been blocked, but it is better and more safely done in a quiet interval phase.

The choice of operation after control of tension will be discussed presently. If, however, medical means fail to lower the pressure, emergency surgery is in order. Exactly how long it is safe to temporize with

nonsurgical therapy is a subject for much debate. If the attack is allowed to continue too long any potentially open angle will become permanently obstructed. It is possible that the use of local corticoadrenal steroid therapy during an acute episode may, by lessening edema and inflammation, delay the formation of peripheral anterior synechias. This theoretic possibility is virtually impossible to confirm or disprove.

Selection of operation in the face of irreducible tension is difficult at best.* If the attack is of considerable duration or there is a history of previous acute attacks, it is safe to assume that there will be more or less angle closure of a lasting nature and proceed on this premise. If the attack is of short duration without previous history, it may be safe to assume, in spite of the failure to respond to drugs, that much of the angle is capable of reopening. It is so unfortunate that here, where we would particularly like the assistance of gonioscopy and outflow estimates, they fail us. When in doubt, it would seem to be safer and more conservative to employ the least extensive operation which might be appropriate rather than to risk unnecessary extra surgical trauma under such precarious conditions.

As a general rule it seems wise to continue medical therapy as long as the tension is falling but to abandon it when the tonometer reading becomes stabilized or has failed to respond. Failure to respond at all calls for that sixth sense, "clinical judgement," in deciding how long to persevere. Some surgeons feel that as much as 12 hours of high tension is dangerously damaging, others are sanguine about at least 48 hours' delay. I must confess that there are so many variable factors to consider that I cannot give any arbitrary time limit.

Medical reduction of tension in an acute

angle block cannot be considered a cure but only a preparation for definitive surgery. This is one glaucoma that we can cure, but the cure is surgical. Continuance of nonsurgical treatment beyond a short preoperative period is not to be countenanced except in the very feeble, aged patient with short life expectancy. Particularly to be avoided is the prolonged use of carbonic anhydrase inhibitors which may hold the tension down without relieving the angle block and thereby allow a salvagable filtration area to become hopelessly encumbered. So, too, may the acetazolamide agents give us a false sense of security and warp the surgeon's judgment. This is particularly true if he tends to base his selection of operation on the result of medical treatment of the acute episode.

Assuming, then, that the acute phase has been controlled, or cannot be controlled, how do we proceed logically? It may be worthwhile to look at a simplified scheme of the course of the disease (fig. 1). With each acute attack the angle suffers some damage and the pressure-regulating outflow is impaired. If the damage is so slight that pressure may be regulated at a level within normal limits, the only requirement is a procedure which will prevent further attacks of angle blockade. In fact, such a step is an essential part of all procedures at any level. If the pressure after one or more attacks levels off just a bit above normal, indicating that the residual angle mechanism requires a small supplement, then minor accessory outflow channels must be created. Proportionately, as the stabilized pressure returns to higher levels, greater accessory outflow is needed. Such situations are indicated in the schematic course shown.

If we could be perfectly sure of our tonometry, if we could always compensate exactly for changes in scleral rigidity, if we had some way of knowing just how much pressure each individual eye could stand, then tonometry after control of an attack would tell us how to operate. Unfortunately,

* Gonioscopy at the operating table, as discussed, since this lecture was given, by R. N. Shaffer and P. A. Chandler before the American Ophthalmological Society, May 30, 1957, may be of considerable value in this situation.

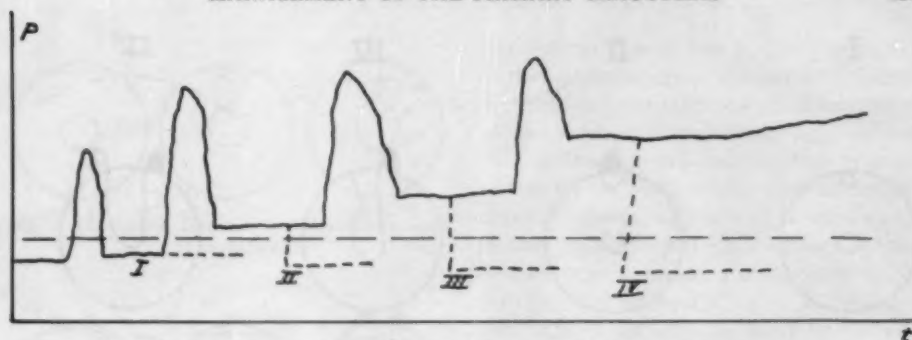


Fig. 1 (McLean). Simplified scheme of course of angle-block glaucoma.

this criterion alone is not so precise. It tells us the extremes but fails to make the finer separations. Therefore I would propose that we use a composite of tonometry, gonioscopy, tonography, ophthalmoscopy, and perimetry to divide angle-closure glaucoma into four steps (table 1).

If the base pressure is normal, if gonioscopy shows an unencumbered angle, if outflow is within normal limits, if the disc is intact, and if the field is full, the case falls into Class I. When all of these factors are borderline or nearly normal it falls into Class II. Similarly, moderate impairment throws it into Class III and severe involvement into Class IV. Each individual surgeon, or institution, will have to fill in, in this table, his own values for normal pressure and outflow according to his own practices and conversion tables.

Figure 2 shows schematically the surgical procedures which I would suggest for these four classes. In Class I where the outflow mechanism has been spared all that is required is adequate communication through the base of the iris. Of course the classical full iridectomy is perfectly applicable but it is not shown. Simple peripheral iridectomy is just as effective, much less disfiguring, and slightly safer. It also gives a little more efficient sphincter mechanism in case miosis should ever be needed for some future reason. However, if peripheral iridectomy is properly selected and adequately performed, it should result in a lasting cure. Peripheral

iridotomy, illustrated below, is just as effective and the choice of some surgeons. Iridodialysis, shown below this, is also as effective but less often used because of technical difficulties. In all of these techniques slow and careful incision with tight wound closure are important lest postoperative flat chamber further compromise the angle. I personally prefer a moderate amount of air to reform the chamber at the end of operation but some other surgeons feel that they got along as well without it. When iridectomy, or its equivalent, is to be done for an uncontrolled acute attack there may be some doubt as to its permanent adequacy. Under these circumstances it may be wiser to place the incision where it will not interfere with subsequent operation.

For Class II peripheral iridectomy would be almost adequate. Just a little supplementary drainage is needed to compensate for the minor impairment of the normal outflow channels. A clumsily done broad iridectomy that results in a minor filtering scar would probably suffice. I would suggest that we achieve this objective through a periph-

TABLE 1
CLASSES OF ANGLE-CLOSURE GLAUCOMA

	I	II	III	IV
Base pressure	N	±	+	+++
Synechias	O	Few	Many	Total
Outflow	N	±	-	Low
Cupping	O	±	+	+++
Field loss	O	Slight	Moderate	Marked

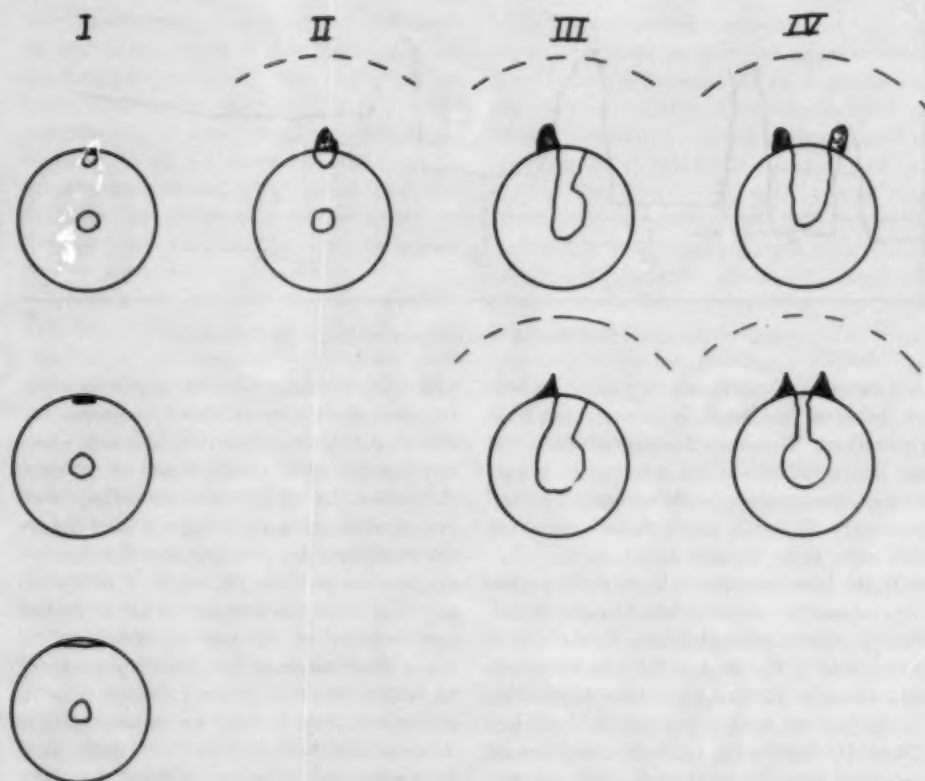


Fig. 2 (McLean). Diagram of surgical procedures in angle-block glaucoma.

eral iridencleisis. Under the usual flap, operation is started as if we were doing a peripheral iridectomy. Two sides of a triangular peripheral iridectomy are cut leaving the base at the iris root attached. This iris flap is then folded back and incarcerated in the wound, the chamber reformed with air, and the conjunctival flap tightly closed. Results with this technique in Class II cases have been most satisfactory.

In more advanced disease, as in Class III, transiris communication is still needed to prevent further attacks plus greater drainage to make up for the more extensive outflow deficit. Here, I would suggest a single pillar iridencleisis and, in the most severe involvement of Class IV, a double pillar iris inclusion. In very severe cases of Class IV

some surgeons may wish to add partial excision of the scleral lip by scissors, punch, or knife. Personally I do not find this necessary if two pillars are well incarcerated at the extremities of a wide incision.

The two lower sketches under III and IV (fig. 2) illustrate a somewhat unorthodox iridencleisis technique which may be applied to either single or double pillar inclusions. Instead of withdrawing the iris, splitting it radially, and incarcerating the pupillary border, a deliberate iridodialysis is performed, the iris is split radially, and the now free root is engaged in the incision. This may be performed in either single (fig. 3) or double (fig. 4) pillar fashion. The slightly increased difficulty of performance is rewarded by a better centralization and less

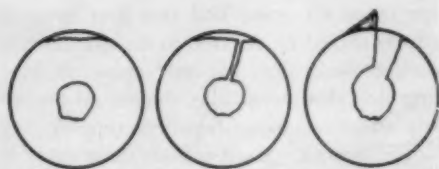


Fig. 3 (McLean). Single-pillar basal iridencleisis (schematic).

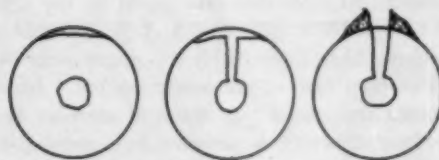


Fig. 4 (McLean). Double-pillar basal iridencleisis (schematic).

updrawing of the pupil, less dazzling from the narrower coloboma, and less resultant astigmatism.

Minimal upward displacement of the pupil is particularly desirable if one is contemplating a later cataract extraction or if the patient has naturally narrow palpebral fissures. I have been using this basal iridencleisis for only a few years and have not used it in most severe Class IV cases so that I do not have enough long-term follow-up cases to be sure that it gives as good and as lasting drainage as the classical technique. However, as far as I have gone, outflow studies and clinical course indicate that it will do as well.

There are two questions that are bound to arise at this point. What about other operations, such as the various sclerectomies (including trephining), cyclodialysis, and the ciliary-destroying procedures (cyclodiatomy and cycloelectrolysis and retrociliary diathermy)? If double pillar iridencleisis is best for severe advanced angle-block glaucoma why not use it in all cases and be sure?

The reasons for avoiding anterior sclerectomies of the trephining type are to be seen in the studies of complications and failures of these operations.^{16,17} Lens injury at the time of surgery,¹⁸ prolonged "quiet iritis,"

and incarceration of lens or ciliary processes in the surgical outflow channel with failure of filtration, plus other complications, seem to me to militate overwhelmingly against these methods. I have no objection to small scleral lip excisions at the time of iridencleisis. Cyclodialysis alone is unsatisfactory because it fails to provide insurance against further angle blockade. Furthermore, cyclodialysis, whether used alone or in combination with iridectomy, is likely to become sealed in these narrow angles and shallow chambers. The ciliary-damaging operations do not attack the primary problem in angle-closure glaucoma. They allow attacks to continue to recur while unnecessarily depressing aqueous formation in the interval stages. The same features that contraindicate prolonged acetazolamide therapy in these patients hold as objections to this surgical approach.

The use of a maximal filtering operation at all stages of angle-block glaucoma is contrary to the whole concept of graded surgical treatment. The idea is to insure against further blockage and to add just enough additional outflow to compensate for the existing deficit. As long as recurring attacks are prevented, the outflow defect should be stationary and surgical supply of the necessary additional drainage should produce a lasting compensatory mechanism.

If we return to the diagram in Figure 1 we see that the dotted lines and Roman numerals reflect the amount of permanent deficit existing in each of the classes and indicate the amount of correction required. Instead of plotting pressure versus time, the same diagram could have shown, using appropriate co-ordinates, outflow resistance versus time, or proportion of angle obstruction versus time.

Overcorrection beyond the requirements of the eye may work out in one of three ways. If our operation is very successful hypotony will be the result with accelerated lens clouding, possible disc and retinal edema, and even detachment of the retina. We then

find ourselves trying to explain to the patient how a successful operation has reduced his sight, making heroic attempts to destroy or diminish the filtering bleb which we have just created, or even operating for cataract or detached retina. If we are more fortunate, delayed reformation of the anterior chamber may so embarrass the angle as to create more severe disease and make the relatively good eye bad enough to neutralize our surgical overendeavor. I am sure that in the past nature has helped me by this mechanism more than once.

If we are very unfortunate, good angle drainage plus good surgical drainage may combine to keep the chamber empty and initiate that most dreaded of postoperative sequences, so-called "malignant" glaucoma. I am sure that I do not understand the entire mechanism of "malignant" glaucoma but I do believe that overoperation in angle-block glaucoma is one way to set it off. We are indeed fortunate that its incidence is relatively low.

What I have been describing thus far is the classical acute angle-block glaucoma. What of the subacute form and what about the fellow eye? I would suggest to you that the subacute form is basically the same disease and that the same principles apply. It may be more insidious in its relative paucity of severe symptoms and therefore more dangerous to the patient who is less urgently compelled to seek relief, but its recognition, evaluation, and surgical, always surgical, therapy follow the same lines.

In every case of angle-closure glaucoma the fellow eye merits careful consideration. With few exceptions it will be subject to the same disease process if, indeed, it has not already been affected. Here we have an opportunity for ideal prophylactic surgery. If the eye is still undamaged, properly performed peripheral iridectomy, or its equivalent, is a perfect preventive measure. Sometimes, as Chandler¹⁹ has pointed out, the fellow eye has such good visual potential, but is in so precarious a state, while the involved

eye is so far gone, that our first surgical efforts should be directed to the eye that the patient thinks does not need attention. Putting this idea across may require an unusually effective physician-patient rapport.

The few exceptions include those cases in which the same anatomic situation does not exist in the second eye. It is theoretically possible that a patient might have two such dissimilar eyes that one would be the site of angle-block glaucoma and the other not subject to it. Conceivably this might occur in a marked case of anisometropia but I have never recognized nor heard of such an instance. Posner²⁰ has shown how peculiarly situated deep peripheral iris crypts may occasionally act as prophylactic physiologic peripheral iridectomies and prevent angle closure. I am able to corroborate his findings. I do not believe however that the mere discovery of such anomalous potential safety valves is sufficient. I am not satisfied until combined mydriatic and dark-room provocative testing have demonstrated their efficacy.

If this be the management of angle-block glaucoma, how then do we approach the open-angle disease? While we do not know the exact cause of the pressure imbalance, tonography and gonioscopy clearly indicate that, in all but a few cases of hypersecretion,²¹ the basic defect is a reduction of outflow capacity and that the site of the obstruction is downstream from the angle. It is probably also beyond the trabecula itself though how far beyond remains to be clarified.

Continued studies by such investigators as Ascher,²² Ashton,²³ Grant,¹² and Theobald²⁴ promise more accurately to pinpoint the location and nature of the increased resistance. In the meantime, we must deal with this, the commonest of the primary glaucomas, in accordance with our best available knowledge and techniques.

If we look at a diagrammatic representation of the course of this disease (fig. 5) we see a slow steady rise with the passage of time in average intraocular pressure, neglecting for the diagram but not forgetting the

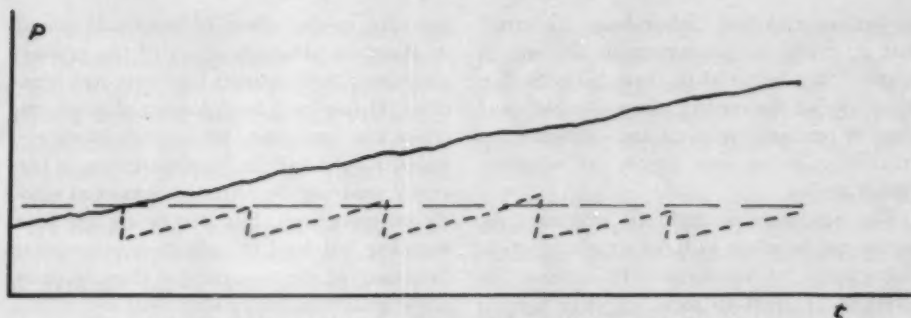


Fig. 5 (McLean). Simplified scheme of course of open-angle glaucoma.

vagaries of diurnal and other variations. Again, as in the scheme for angle-block glaucoma, we might as well have plotted outflow resistance against time, but not synechias, for here they are not the problem. The overall course of the disease is one of slow but inexorable decrease in outflow facility and rise in pressure with the attendant damage to nerve and retina, field and vision.

Our first step in management must be early recognition; more routine tonometry, perimetry, ophthalmoscopy, and vision testing, not just on our part, but by those who are doing routine physical examinations, "health" examinations, and industrial surveys. Simple screening techniques can be taught and introduced into the nonophthalmologic areas of medicine. Those who are practicing preventive medicine are in the habit of doing routine blood tests, X rays, and the like which have a much lower yield than the approximate two percent²⁵ to be expected from routine glaucoma screening in and beyond middle age.

The best area for such missionary work is among our medical students. For corroboration of borderline diagnoses, Becker's²⁶ quotient $P_o/C < 100$ (tonometer reading \div coefficient of outflow < 100) is very useful to add to our regular battery of tests, as is the tonographic water provocative test.²⁷

Assuming that the cases are found, we must next plan their control. Since the outflow resistance and tension steadily mount, the problem is much more difficult

than that presented by angle-block glaucoma. No single operative intervention, no single medical schedule can be counted upon to give lasting results. Obviously any step which will be adequate far along in the disease (the right-hand end of fig. 5) would be excessive in the early stages.

This statement is even more valid for surgical measures than drug therapy. Successfully performed overoperation will surely produce hypotony, as most of us have had the misfortune to see. Overdosage with miotics is less damaging and easier to control. It can be readily demonstrated tonographically that the major effect of miotic therapy is by increasing facility of outflow. There seems to be a limit beyond which this increase will not go so that overdosage is not risky. It is, however, undesirable and also subjectively unpleasant. We would prefer to manage the disease in step-wise fashion dropping the pressure level and outflow resistance to reasonable levels again and again as the severity of the disease increases. This I have attempted to indicate in the dotted lines. The repeated rises are shown, deliberately, as reaching or just exceeding the limit of normal, for this is how they usually run. The ideal would be to add each therapeutic increment just short of this level.

What are these therapeutic steps and how are they applied? I had planned to review with you a whole host of old and newer drugs but I shall not. Your own Dr. Leopold has so ably covered them in his recent and

as yet unpublished Schoenberg Lecture²⁸ that it would be presumptuous for me to reiterate this material in these halls. Suffice it to say that the control of open-angle glaucoma is primarily medical and becoming increasingly so as our supply of effective agents grows.

The first steps in my little schematic diagram can be taken with the simpler miotics. Subsequent steps represent increase in strength or shift to more effective agents. Still later steps are addition of aqueous-inhibiting agents. Finally, but not before, in reliable, observable patients, comes surgical enhancement of outflow or surgical suppression of inflow. This major step usually allows us to drop our medical adjuvants back to zero or nearly so. We may then pick up again as needed with nonsurgical steps until once more they are exhausted and further operation is in order. We can take some comfort in the evidence, scanty though it may be, that long-standing open-angle glaucoma eventually tends to burn itself out, at least as evidenced by a lessening of its rate of progress.²⁹

All of this applies to the ideal patient. In the unco-operative, unreliable individual, early operation, undesirable as it may be on theoretic grounds, is the only practical solution.

This step-by-step control of open-angle glaucoma seems to me to be the most logical approach but it is by no means easy of execution. It requires very careful tailoring of the treatment to fit each individual. It means study of diurnal variations in each patient for proper timing of his medication. It is not sufficient to have the disease under control at the time of the office or clinic visit but out of bounds at other times of day or night. There is less load on the ocular plumbing if caffeine, nicotine, and fluid intakes are controlled. I have no evidence on which to limit reasonable alcohol intake or scotopic visual activity in open-angle glaucoma.

Emotional disturbances are yet another factor. Ripley and Wolff³⁰ have shown the

pressure raising effect of emotional stimuli in glaucoma although which of the primary glaucomas their patients may have had is not clear. It has long been known that psychic crises can precipitate acute angle-block episodes. Kaufman,³¹ in our department, is currently studying the effect of emotional activity on tonograms. His studies are far from complete but tend to indicate a pronounced flattening of the tonographic slope in open-angle glaucoma under emotional stress. The responses are rather clear cut and surprisingly prompt. How much of this is mediated through hemodynamics must still be clarified. Gonioscopy and pupillary control make it clear that the effect is not a "Cannon cat" mydriasis. In any event experimental evidence and clinical experience both tend to support the use of sedatives, tranquilizers, and even psychotherapy in the management of emotionally labile patients with open-angle glaucoma.

What criteria shall we use for the adequacy of our management of this disease? Many would set an arbitrary level of tonometer reading as a goal, usually without even bothering to consider ocular rigidity or other inherent errors of instrumentation. Recently a few have swung over to tonographic control, again using arbitrary levels. I would suggest, along with the more conservative group, that vision and visual field are what we are trying to conserve and that sensitive perimetry should be the ultimate criterion; this means tangent screen fields by the most delicate technique which is reliable in the individual patient. The earlier the disease the more important are small test objects and reduced illumination.

I do not believe for a minute that any one set of conditions can be standardized for all cases but we must standardize our techniques for adequate comparison at different times in the same patient. A word of caution is due here to recall to our minds the misleading changes wrought by alterations in transparency of the media or pupillary size. By the use of individually standardized sensi-

tive perimetry we may be able to establish for the individual patient a fairly accurate critical level of tonometer reading or perhaps of outflow coefficient beyond which that person cannot safely go. I would, nevertheless, adhere to his fields as the patient's best guide and ultimate concern.

As Reese³² has shown and we have confirmed, there is a relationship between level of systemic blood pressure and tolerable intraocular pressure. Furthermore, I have seen a patient lose field at the same tension level on which he previously maintained it when his internist succeeded in drastically controlling his hypertension. All of this occurred in spite of theoretic beneficial effects on the eye from the systemic regime.

When medical control has reached its practical limit either in drug efficacy or patient co-operation, surgery is in order. Can we be as arbitrary in selecting techniques as we were in angle-block glaucoma? I do not believe so. Some day we may be. It is clear that nothing can be expected from iridectomy. Since the basic defect is in outflow it would seem most reasonable to approach operations that increase outflow first. Any good filtering operation, external or internal, is suitable.

In my own experience corneoscleral trephination is the most reliably effective over the longest interval when the anatomic conditions are favorable. This implies a wide angle at the selected site. Projection of iris, lens, or ciliary tissue may impair its long-term result in open-angle disease when the angle space was not adequate.

Therefore, for open-angle glaucoma, in an angle that is open but none too wide, I would suggest some other form of anterior sclerectomy or preferably iridencleisis.

Iridencleisis, which would be my second choice in a wide open angle and my first choice in a narrow open angle, is to be avoided if the iris tissue is atrophic or fibrotic. Cyclodialysis, which would be my first choice in aphakic open-angle glaucoma, has a chance of remaining effective which

varies directly with the chamber depth at its periphery. There are many modifications of these operations which have merit under varying circumstances. I have neither the time nor the ability to give them all their full due.

The attack from the rear, attempting to balance outflow deficit by induced inflow reduction, does not appeal to me as much on theoretic grounds. I must confess that my lack of enthusiasm for cyclodiathermy, cycloelectrolysis, and retrociliary diathermy is due in part to my own poor results with these three operations. Others^{33,34} report better results and some who deal with much Negro glaucoma³⁵ consider them the primary operations of choice. In this race, with which my experience of recent years has been very limited, filtering operations do seem to fare unusually poorly in spite of attempts to maintain drainage by radiation, steroid therapy, and other means. Perhaps these patients do deserve a more extensive trial of ciliary body surgery.

The combination of angle-block and open-angle mechanism is either very rare or very rarely recognized. It can be recognized more readily when open-angle disease is clearly diagnosed first, in a patient with narrow angles which later begin to block. When there is evidence that angle closure has occurred one or more times before the patient is first seen, the diagnosis rests on assessment of the disproportion between angle encumbrance on the one hand and coefficient of outflow, tension level, field loss, and cupping on the other. If a "cured" early angle-closure glaucoma maintains a steady rise—not just a steady elevation—in pressure level with a steady decrease in out-flow facility, it must be assumed that the open-angle mechanism is also operating. Management of these cases would seem to be early operation for the angle blockage, selected on an assessment of the over-all situation at the time in terms of existing deficit as if it were all of that type, coupled with subsequent control on the principles of pure open-angle disease.

Selection of medical and surgical methods for both of these factors have already been discussed.

In conclusion, let me remind you again at the risk of being trite that, in spite of the mis-

leading wording of my title, we should not be technicians managing the glaucomas, but physicians managing patients with glaucoma.

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VITREOUS CHANGES AND THE MECHANISM OF RETINAL DETACHMENT*

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New York

Since we studied the anatomy of the periphery of the retina and the pathogenesis of retinal holes,¹⁻³ we have seen 20 more eyes which had perforated round holes, but which did not develop retinal detachment. Adams⁴ recently reported observations similar to ours. From his description, we believe that these holes are probably the result of congenital rosettes. Retinal holes without subsequent retinal detachment have been reported by Arruga,⁵ Vogt,⁶ Sabbatini,⁷ Jeandelize and Baudot,⁸ Burch,⁹ Genet,¹⁰ Guillet,¹¹ Trantus,¹² Hanssen,¹³ and Knapp.¹⁴ The same thing was very recently reported by Colyear and Pischel,¹⁵ who also reviewed the literature very thoroughly. Most of these reports are based on clinical observation rather than histologic study. Some of their cases could be explained by the presence of chorioretinal adhesion but in most cases there was no apparent reason why a retinal detachment did not occur.

The role of vitreous changes as a possible contributing factor in the pathogenesis of retinal detachment was especially stressed by Knapp. Liquefaction of vitreous is generally accepted as an important factor. Von Sallmann and Reiger¹⁶ confirmed this in every one of their cases. Gonin¹⁷ described the shrinkage of the vitreous and his findings were confirmed by Lister,¹⁸ Lindner,²² von Sallmann, and others. Von Sallmann and Reiger, Lindner, Vogt, and Amsler¹⁹ all observed detachments of the vitreous. It is our belief, on the basis of our study of simple, uncomplicated anatomic material, that shrinkage of vitreous and detachment of vit-

reous are essentially the same type of vitreous change.

Recently this type of vitreous change has been stressed by Pischel.²⁰ He reports that he observed it in more than 90 percent of his retinal detachment cases.

Another important factor now being stressed in its relation to retinal detachment is vitreo-retinal adhesion. Leber²¹ observed that rupture of the retina occurs at a place where there is an adhesion between the vitreous and the retina. This observation was seconded by Gonin,¹⁷ Lindner,²² Boeck,²³ and Wadsworth.²⁴

Our present report on the morphology of vitreous changes confirms the belief that the two main anatomic factors in the etiology of the idiopathic type of retinal detachment are liquefaction of the vitreous and vitreo-retinal adhesion.

METHOD OF STUDY

In the Eye-Bank library we have preserved "normal eyes" which were used for corneal grafting, then fixed in Bouin's solution, and kept in 80-percent alcohol.

Our first step in this study was to estimate the extent of post-mortem changes in the vitreous of eyes treated according to normal Eye-Bank histologic routine. Eyes are usually put in fixative within two days and rarely over three days after the death of the donor. To estimate the post-mortem changes we used 40 fresh beef eyes, dividing them into groups of four, keeping them in the refrigerator for from one to 10 days. After their specified time in the refrigerator, the eyes were fixed in Bouin's solution, then dehydrated with alcohol in the routine manner, and examined. We found that within five days there was no visible morphologic change in the vitreous. After six days there was some precipitate at the surface of the vitreous which might

*From the laboratory of The Eye-Bank for Sight Restoration, Inc., Manhattan Eye, Ear, and Throat Hospital, under grants B153 and B1130 from the U. S. Public Health Service. This material was presented in exhibit form before the 1955 annual convention of the American Academy of Ophthalmology and Otolaryngology, Chicago.

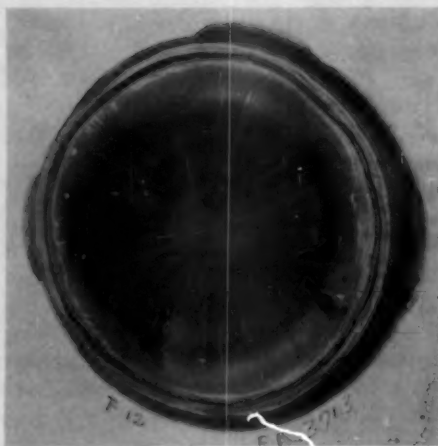


Fig. 1 (Teng and Chi). Normal vitreous (equatorial section).



Fig. 2 (Teng and Chi). Mild liquefaction of vitreous at the central core (Type 1).

have been the result of autolytic changes in the retina.

Secondly, we compared the appearance of fresh vitreous with fixed specimens, in order to evaluate the extent of changes due to fixation.

We took 46 fresh human eyes, obtained immediately after removal of the cornea for grafting, and, without placing them in fixative, we removed the remaining cornea, iris, and lens. We then suspended the eye in normal saline or in 1.2-percent hypertonic saline and examined the eyes with the slitlamp. Because of the hydrophilic property of vitreous, we worked as quickly as possible; the examination was usually made within 30 minutes.

We found that the appearance of liquefaction of the vitreous was very similar to the slitlamp appearance in living eyes and very comparable to the fixed specimen.

Among our preserved specimens, we have found that the vitreous of all babies' eyes and most young persons' eyes is without liquefaction. We concluded, then, that the liquefaction we found in these supposedly normal adult eyes was due to age rather than post-mortem change, or artefact.

We found Bouin's solution a very satisfac-

tory fixative for our purpose. It renders tissue and solid vitreous harder, more opaque, and yellowish in color, which makes examination easier.

OBSERVATIONS

I. SENILE LIQUEFACTION OF THE VITREOUS

A. Type 1 (figs. 2 to 6 inclusive)

Our study of liquefaction of vitreous showed that this is a very common phenomenon in the older age groups. Sometimes, however, it may start as early as the age of 26 years, although, on the other hand, we had one specimen which exhibited very little liquefaction at the age of 87 years. This type of liquefaction is most common, being found in 35.3 percent of the eyes examined (tables 1 and 2).

It usually starts in the central portion of the vitreous and extends more in the anterior-posterior direction. The anterior periphery is less involved, the major portion of the vitreous change being around the central core and slightly posterior to the center.

Although this type of liquefaction can be



Fig. 3 (Teng and Chi). Moderate liquefaction at center. Fairly thick solid vitreous at the periphery (Type I).

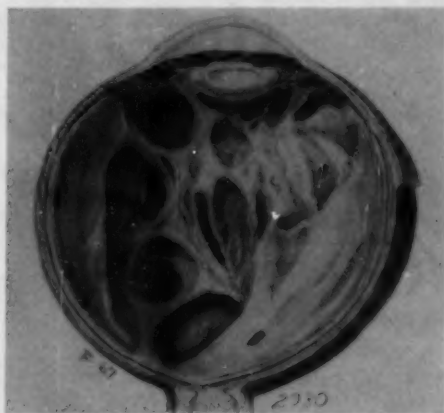


Fig. 4 (Teng and Chi). Moderate liquefaction at the center. Anterior hyaloid membrane is disturbed (artefact). Common type (Type I).

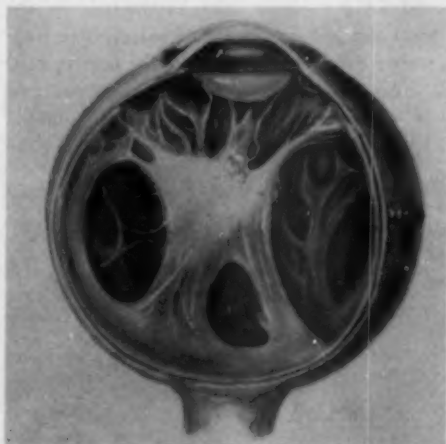


Fig. 5 (Teng and Chi). Moderate liquefaction, "condensation" of vitreous at center. Unusual type (Type I).



Fig. 6 (Teng and Chi). Severe liquefaction, with loss of substance. Hyaloid membrane remains intact. Rare type (Type I).

very irregular in location and degree, sometimes forming irregular pockets, the anterior and peripheral parts are usually least involved. These portions form a solid shell of vitreous which protects the periphery of the retina. We also noted that liquefaction is often more extensive over the upper portion of the vitreous, probably due to gravity.

B. *Type II* (figs. 7 to 10 inclusive)

The second, less common (table 2), type of liquefaction is distinguished by anterior contracture of the solid part of the vitreous, leaving the posterior part liquid. This condition may be described as posterior vitreous detachment, posterior hyaloid membrane de-

TABLE 1
STATISTICAL STUDY OF LIQUEFACTION OF VITREOUS

Age	Normal	Type I		Type II		Total
		Mild	Severe	Mild	Severe	
0-10	14	—	—	—	—	14
11-20	3	—	—	—	—	3
21-30	3	1	—	—	—	4
31-40	10	4	1	—	—	15
41-50	19	10	8	1	—	38
51-60	19	8	14	4	5	50
61-70	15	7	17	—	13	52
71-80	3	2	6	2	5	18
81-90	3	—	2	—	7	12
Unknown	5	1	1	2	17	26
TOTALS	94	33	49	9	47	232

TABLE 2
TYPES OF LIQUEFACTION IN 232 EYES STUDIED

	No. of Eyes	Percent	
Normal	94	40.5	
Type I Liquefaction	Mild 33	14.2	35.3
	Severe 49	21.1	
Type II Liquefaction	Mild 9	3.9	24.2
	Severe 47	20.3	

tachment, or anterior contracture of the vitreous. We refer to it as Type II liquefaction of vitreous.

This kind of change may have several re-

sults. It may occur without disinsertion of the posterior attachment of hyaloid membrane around the optic nervehead, but this is extremely rare. More usually there is posterior detachment. The contracture of the solid vitreous may vary in degree. In the worst cases it may be called massive contracture of the vitreous. The liquid part is usually not coagulated in the fixation process, but occasionally it may become coagulated (fig. 10), probably due to an increase in the protein content in the liquid vitreous.



Fig. 7 (Teng and Chi). Mild liquefaction from behind with anterior "contracture" or posterior detachment (Type II).



Fig. 8 (Teng and Chi). Moderate liquefaction without posterior disinsertion. Rare type. Usually there is posterior disinsertion (Type II).

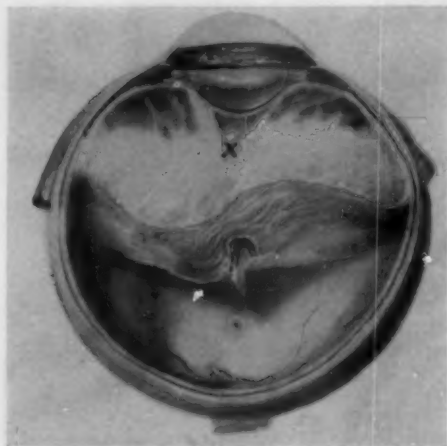


Fig. 9 (Teng and Chi). Moderate liquefaction. Tear occurred at the insertion of the vitreous. Common type (Type II).



Fig. 10 (Teng and Chi). Moderate liquefaction. Liquid vitreous coagulated by fixative. Rare finding (Type II).

II. VITREO-RETINAL ADHESION

A. Senile peripheral vitreo-retinal adhesions (fig. 11)

According to Salzmann,²⁸ vitreous is firmly attached at the region of the ora serrata and to the ciliary epithelium in a zone some 1.5-mm. broad immediately adjacent to the ora serrata. Adhesions between the surface of the vitreous body and the retina in the region anterior to the equator have commonly been observed by Gonin,²⁹ Samuels,³⁰ Lindner,²⁷ Redslob,²⁸ and Schepens.³⁰ Lindner noticed that the shrunken vitreous becomes detached up to the ora serrata and, in older persons, it remains adherent to the anterior part of the retina.

We have made a study of 68 eyes from different age groups. From our analysis of these eyes, we concluded that this type of vitreo-retinal adhesion forms a band around the periphery of the retina. It looks like an extension of the vitreous insertion, backward into the retina. The degree of extension of the peripheral adhesion varies a great deal, but it has a definite tendency to increase with age (chart 1). This kind of vitreo-retinal adhesion is not found in children's eyes and

is rarely present before the age of 30 years. In the younger age group the adhesion is sometimes weak and separable. It may be that these are early adhesions, which will strengthen as the person grows older.

Usually the posterior borderline of the



Fig. 11 (Teng and Chi). Intact hyaloid membrane shows extent of senile vitreo-retinal adhesions.

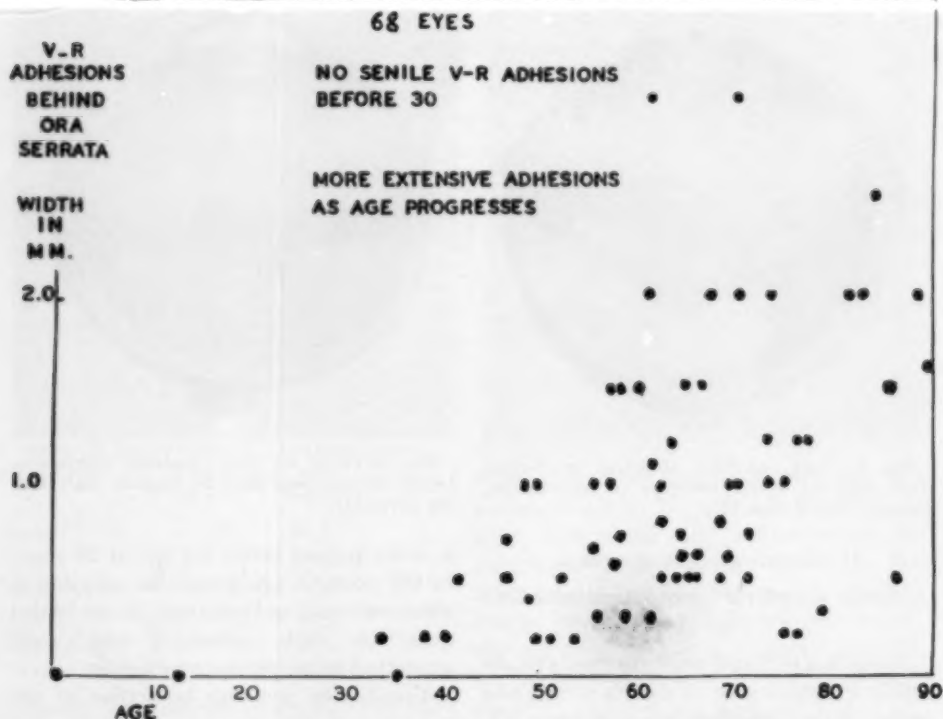


Chart 1 (Teng and Chi). Widths of extension of senile vitreo-retinal adhesions.

adhesion is a rather smooth curved line, running along the extension, varying, perhaps, in different meridians. It is usually widest on the temporal side. Often this senile band type of vitreo-retinal adhesion is combined with local vitreo-retinal adhesions, making the line of adhesion irregular and projecting acutely here and there. This projection is more dangerous than the regular line of adhesion as a cause of retinal detachment. Senile vitreo-retinal adhesion has no relationship to peripheral cystoid degeneration (fig. 11). Peripheral cystoid degeneration can be very extensive when there is only a very mild degree of vitreo-retinal adhesion, and vice versa.

The width of the adhesion varies in different cases from 0.0 to 3.0-mm. posterior to the ora serrata (chart 1). If it is 3.0-mm. wide, that brings the line very close to the equator

of the globe. Histologically it exhibits a union between the hyaloid membrane of the vitreous and the internal limiting membrane of the retina. It seems to be cementlike in nature.

B. Local vitreo-retinal adhesions (figs. 12 to 23 inclusive)

By local vitreo-retinal adhesion, we mean a small spot or patch of the retina which adheres to the vitreous in any part of the eye. So far, in our specimens, we have found that this type of vitreo-retinal adhesion is always due to congenital rosettes and folds in the retina. Hagedoorn and Sieger³⁰ recently reported congenital vitreo-retinal adhesions. We feel that many of these can be explained by rosette formation.

Congenital rosettes and folds (figs. 24, 25, 26, and 27) are due to abnormal persistence

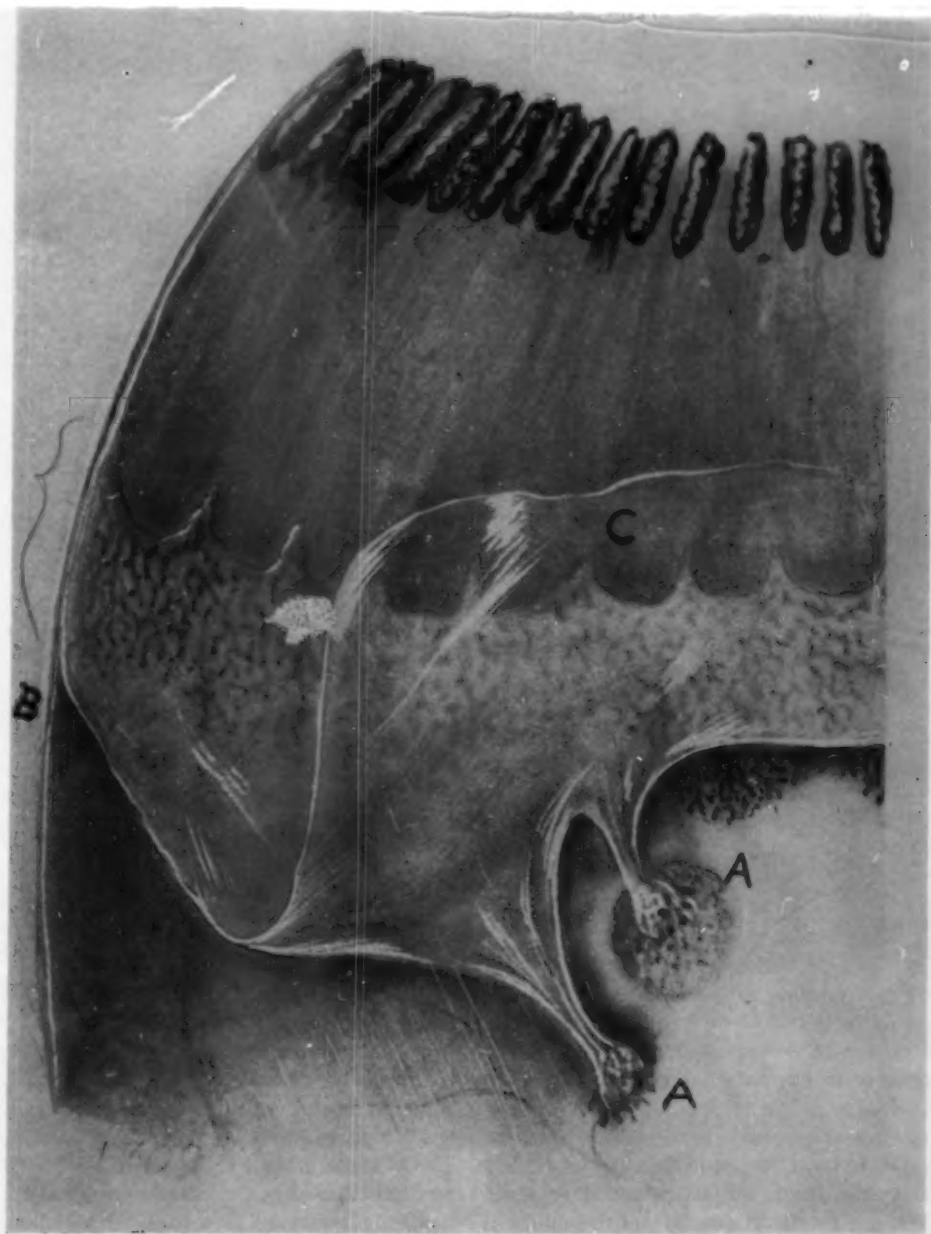


Fig. 12 (Teng and Chi). (A) Local vitreo-retinal adhesion at congenital rosette. (B) Senile vitreo-retinal adhesions. (C) Intact hyaloid membrane.

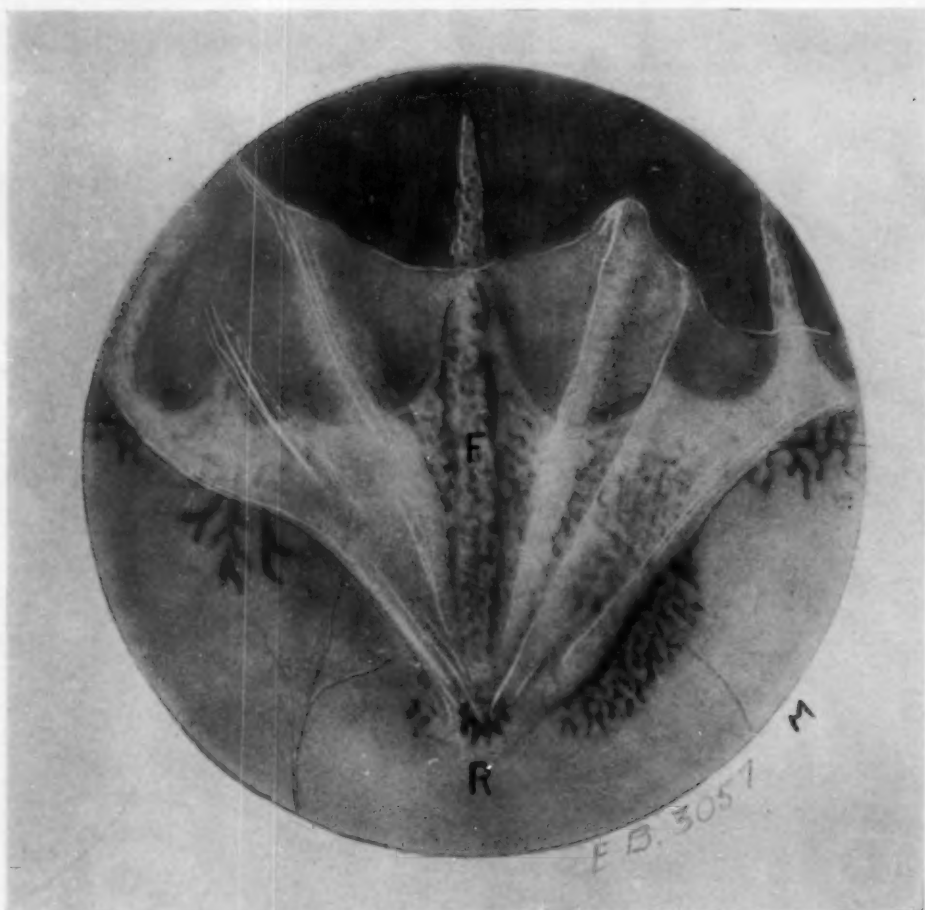


Fig. 13 (Teng and Chi). Local vitreo-retinal adhesions at congenital rosette (R) and fold (F).

of proliferation of neuroepithelium of the retina in the phase of differentiation.³ The area may be very small, involving only a few cells, or it may be a larger mass, differentiated later into a typical rosette or mass of rosettes. They may be detached from the retina to form floaters in the vitreous, or they may stay inside the retina and degenerate. When a rosette is freed from the retina, it may leave a degenerated spot, an operculated hole, or a round hole in the retina. These pre-existing round holes in the retina can be persistent, lasting a lifetime without causing

retinal detachment unless there is Type II liquefaction of the vitreous (fig. 28).

The most important point we want to make is that there is always a local vitreo-retinal adhesion at the rosette, and sometimes a small area of adhesion around the rosette. The adhesion is fibrillar in nature. The fibers are produced by abnormally proliferated neuroepithelium and they form the adhesion to the vitreous. There is usually very little involvement of the pigmented epithelium or the choroid under the area of adhesion. Although this type of congenital defect may



Fig. 14 (Teng and Chi). Local vitreo-retinal adhesion at papillary type rosette.



Fig. 16 (Teng and Chi). Local vitreo-retinal adhesions around holes.

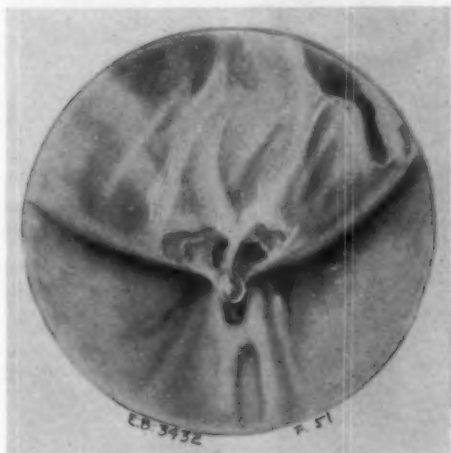


Fig. 15 (Teng and Chi). Local vitreo-retinal adhesion at detaching rosette. Hole with operculum.

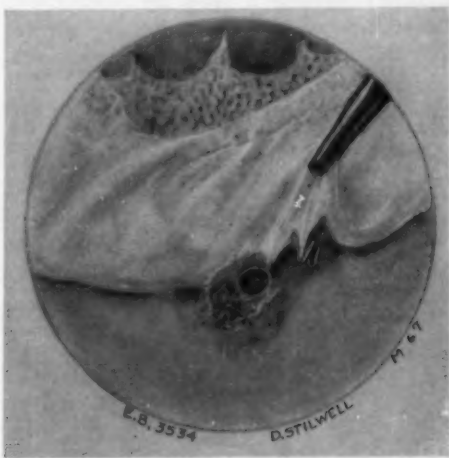


Fig. 17 (Teng and Chi). Local vitreo-retinal adhesion around a large hole (left) and small hole (right). Pulling causes V-shaped tear.

be found in any part of the retina, it is most common in the periphery.

From the standpoint of etiology of retinal detachment, a local vitreo-retinal adhesion is more important than the senile type, because it can occur posterior to the senile type. Then, because it occupies a smaller area, the

force pulling on the retina is more concentrated.

Motion (figs. 29 and 30)

We believe that Type II liquefaction of vitreous and either type of vitreo-retinal adhesion are fundamental anatomic precondi-

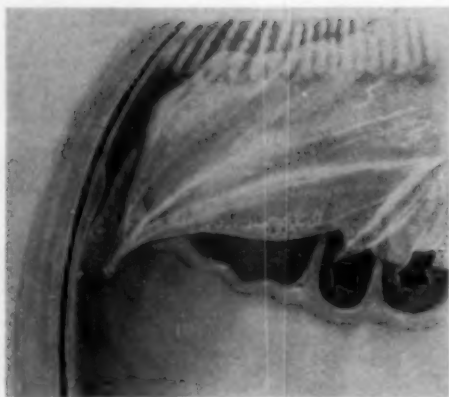


Fig. 18 (Teng and Chi). Baby's eye. Adhesion found only at congenital rosette. Normal insertion of vitreous at pars plana. Traction causes tear at pars plana.

tions of retinal detachment and that motion is the precipitating factor.

Best²¹ emphasized that every movement of the eyeball caused movement of the vitreous and this movement would exert traction at any point of vitreo-retinal adhesion. It is known that liquid and solid media react dif-

ferently to the effect of inertia. In Type II liquefaction of vitreous, there is solid vitreous in front with the hyaloid membrane immediately posterior to the solid mass. Liquid vitreous is behind this. According to the principles of inertia, liquid is more resistant to movement and stoppage than a solid. Therefore, in an eye with Type II liquefaction, a sudden motion or stoppage of motion may create traction at the site of vitreo-retinal adhesion.

The effect of momentum depends upon its



Fig. 19 (Teng and Chi). Long papillary type rosette (A). Vitreo-retinal adhesion around rosette (B).

Fig. 20 (Teng and Chi). Vitreo-retinal and choroid-scleral adhesion. Strong adhesion to the sclera. No danger of retinal detachment.

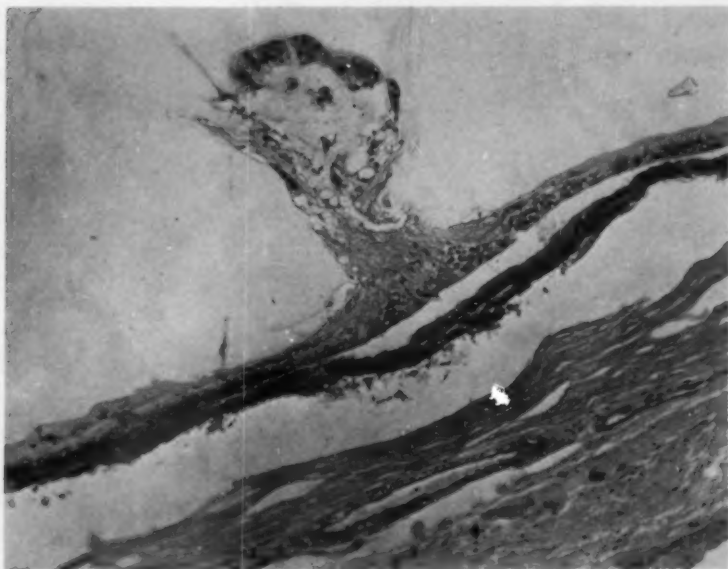


Fig. 21 (Teng and Chi). Section of papillary type rosette with local vitreo-retinal adhesions, fibrillar in nature. (Gross view in Figure 27.)

speed and direction, so that a quick movement may exert a great deal of force at the point of vitreo-retinal adhesion. Motion may be either in a straight line or rotary. If the force which pulls in the direction causing the greatest traction at the point of vitreo-retinal adhesion is strong enough, it will precipitate a tear in the retina, especially if the retina is weakened by degeneration, which is often the case at the site of a rosette.

The tear may be U- or V-shaped, or a long, horizontal tear, depending on the size and extent of the adhesion and the strength and direction of the force. We think that round or U- and V-shaped tears are usually the result of a local vitreo-retinal adhesion and that long horizontal tears are due to senile vitreo-retinal adhesions. In some cases the tear may start from a local adhesion and extend to a senile adhesion to form a long, linear tear.

Once the retina is torn, subsequent motion tends to elevate the retina, allowing the fluid vitreous to migrate behind it and causing separation of the retina. When the retina

is detached, it is much more vulnerable, so that secondary tears or enlargement of the primary tear are very common occurrences.

In this connection, it is worthwhile to note the value of Hilding's²² method of studying the motion of vitreous. Further studies along



Fig. 22 (Teng and Chi). Section of a floater from a detached rosette with fibrillar adhesion to the vitreous.

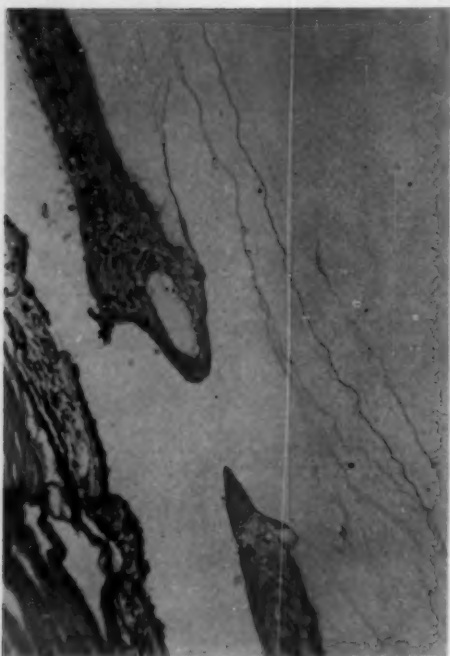


Fig. 23 (Teng and Chi). Section of a perforated retinal hole resulting from a detached rosette. Local vitreo-retinal adhesion at the lip of the hole.



Fig. 24 (Teng and Chi). Appearance of congenital rosettes and folds in retina of baby's eye.

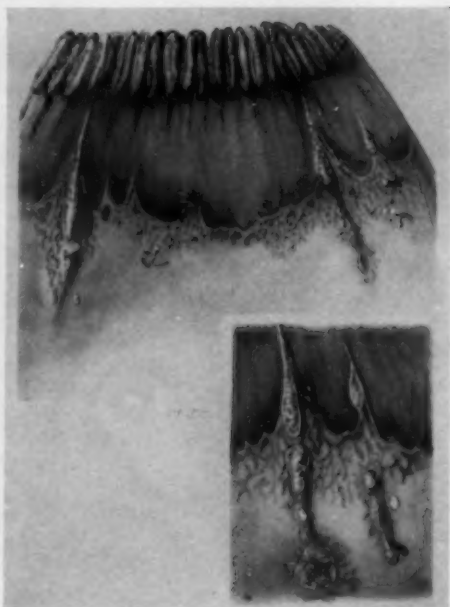


Fig. 25 (Teng and Chi). Appearance of retinal rosettes and folds in eyes of older people.

these lines, bearing in mind the anatomic changes in vitreous, might be very profitable.

Mechanism

Our present study does not add much that is new to the knowledge of the etiology of retinal detachment, but it aims to correlate various factors and thus simplify the picture.

We believe that the three principal factors in retinal detachment are Type II liquefaction of vitreous; vitreo-retinal adhesion, either senile or local; and the precipitating factor—motion.

For convenience of presentation, we divide the idiopathic retinal detachments into three main types: (1) retinal detachment with pre-existing round holes; (2) retinal detachment with U- or V-shaped tears; and (3) post-cataract retinal detachments.

1. *Retinal detachment with pre-existing round hole* (fig. 31). In such cases the vitreous is adherent to the retina at the point of a congenital rosette and anterior to the lip



Fig. 26 (Teng and Chi). Detaching rosettes, one with hole formation.



Fig. 27 (Teng and Chi). Papillary type rosette.

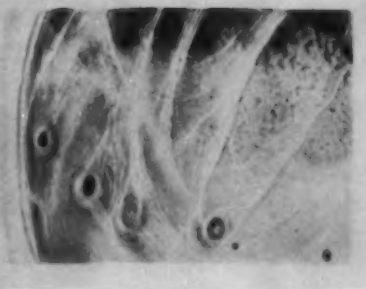
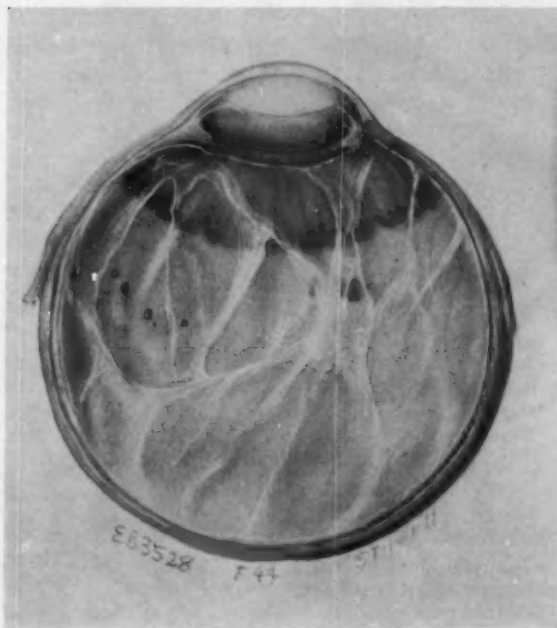


Fig. 28 (Teng and Chi). Multiple perforated holes in retina by hyaloid membrane and layer of solid vitreous.

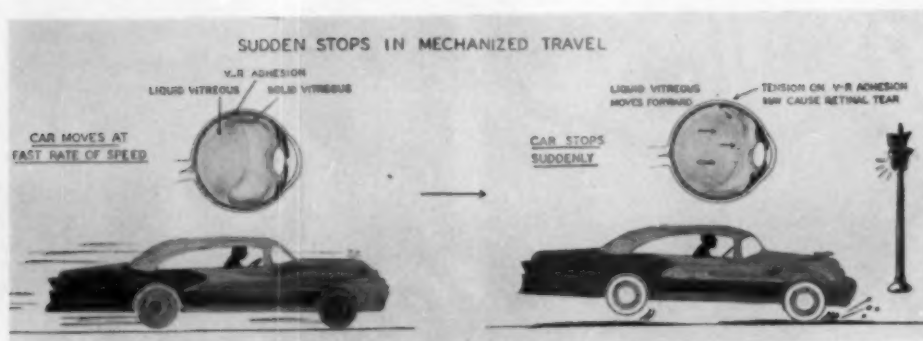


Fig. 29 (Teng and Chi). Diagram illustrating the role of sudden motion in the mechanism of retinal detachment.

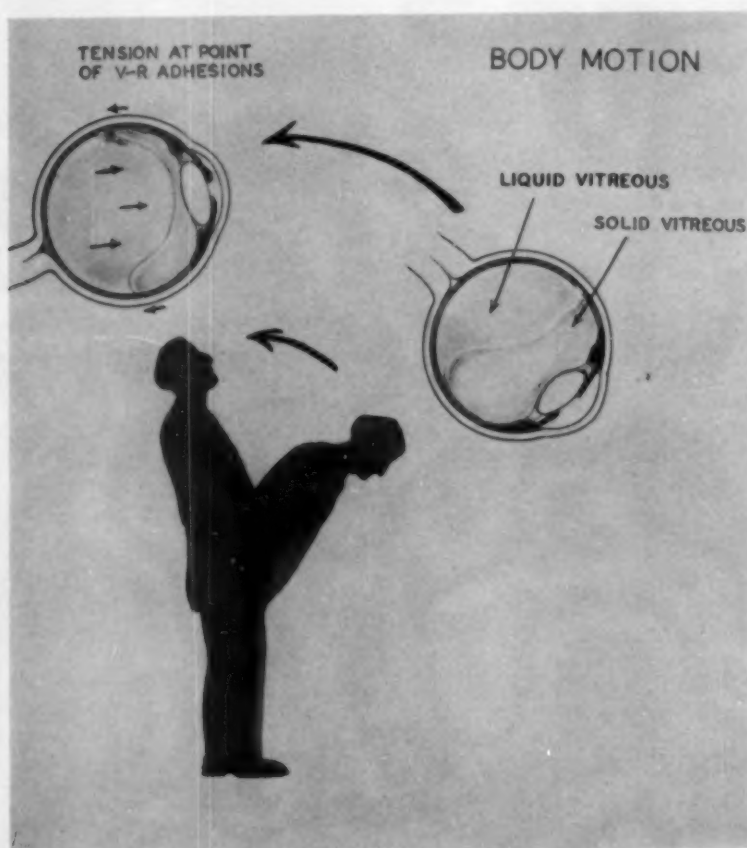


Fig. 30 (Teng and Chi). Diagram illustrating the role of body movements in the mechanism of retinal detachment.

of the hole. If the adhesion occurs without the additional factor of Type II liquefaction, there will be no retinal detachment. If there is Type II liquefaction, however, and the hyaloid membrane becomes detached up to the level of the hole, then any motion will make the liquid vitreous push the solid vitreous forward, creating traction at the site of the adhesion and lifting the retina so that the fluid may enter the subretinal space, through the pre-existing hole. This is more dangerous if the hole is in the posterior sector, especially near the equatorial level, because at this point the motion creates a greater excursion effect.

2. *Retinal detachment with a U- or V-shaped tear* (fig. 32). A U- or V-shaped tear occurs when there is Type II liquefaction plus either local vitreo-retinal adhesion or a combination of senile adhesion with local adhesion. In this case, when the detachment of the hyaloid membrane is up to the level of the line of adhesion, a sudden motion may exert a strain along that line, till it tears at the point where the traction is strongest. Wider adhesions will produce a U-shaped tear, while narrower adhesions produce V-shaped tears. The subsequent lifting effect of traction elevates the retina and allows the liquid vitreous to enter the subretinal space, through the tear. Thus the retina is separated from the pigmented epithelium.

3. *Postcataract retinal detachment* (fig. 33). Postcataract retinal detachments differ from the two previously described types mainly in that the removal of the lens aggravates the condition by allowing the solid vitreous to come further forward in the eye, so that under stress there is even greater traction at the site of the vitreo-retinal adhesion.

CASES FOR ILLUSTRATION

SUBCLINICAL CASES

EB 3883 (fig. 34). The patient was a woman, aged 46 years, who had died of the effects of carcinoma of the uterus. Her eyes were donated to the Eye-Bank. Morphologic

study showed a moderate degree of Type II liquefaction of the vitreous. Over the upper temporal region of the retina, about 3.0 mm. behind the ora serrata, there was a local vitreo-retinal adhesion and several pre-existing round holes. These holes were the result of detached congenital rosettes. There was a U-shaped tear in the retina and at the tip of the anterior lip of the tear was a tiny rosette with a strong vitreo-retinal adhesion pulling at it. This case was an excellent example of a U-shaped tear due to traction at the point of a local vitreo-retinal adhesion.

EB 2452, O.D. (fig. 35). The patient was a 72-year-old woman who had died of a coronarary occlusion. She had had a cataract operation but no further details of the eye history were available. Examination revealed an aphakic eye with early retinal detachment. There was Type II liquefaction of the vitreous. The vitreous was contracted and, because there was no lens to hold it back, it had moved further forward than is normal. At the upper temporal region, 5.0 mm. behind the ora serrata, there were four round holes. There were strong vitreo-retinal adhesions around the holes. There was evidence of strong traction having pulled at the site of the vitreo-retinal adhesion and the retina around the holes was definitely separated, but not very far.

CLINICAL CASES

EB 4087 (fig. 36)* O.D. This case and the history were obtained through the kindness of Dr. George Lane of Plainfield, New Jersey. The patient was a woman, aged 56 years, who had died of pulmonary embolism. On September 25, 1955, Dr. Lane had found bullous separation of the retina at the lower temporal region of the right eye. A diathermy operation was performed the next day. Seven days after the operation the retina was completely reattached. On October 4, 1955, the patient died of pulmonary embolism and the eyes were donated to the Eye-Bank.

* Not in the 1955 exhibit.

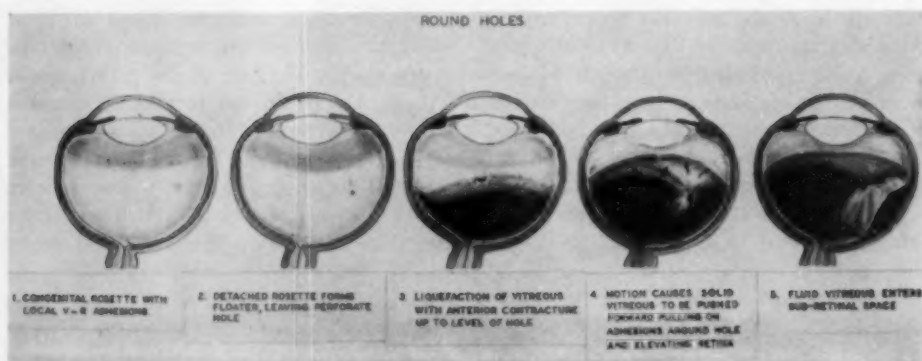


Fig. 31 (Teng and Chi). Round holes.

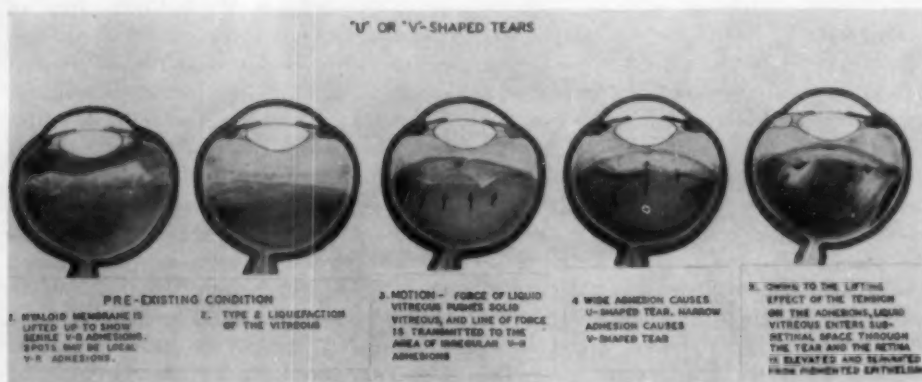


Fig. 32 (Teng and Chi). U-shaped and V-shaped tears.

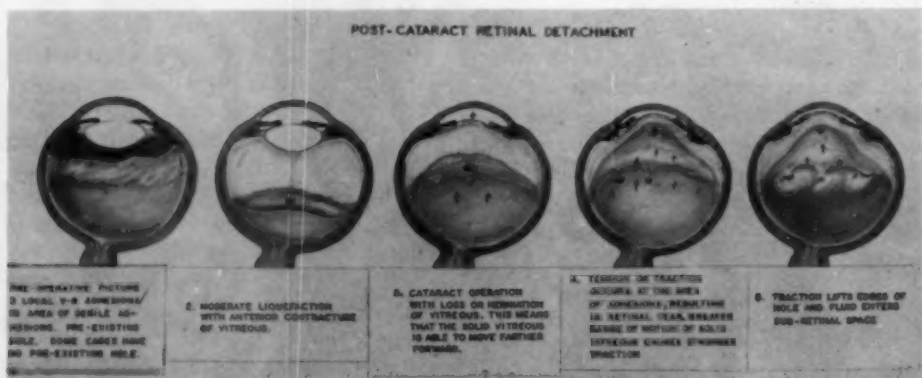


Fig. 33 (Teng and Chi). Postcataract retinal detachment.

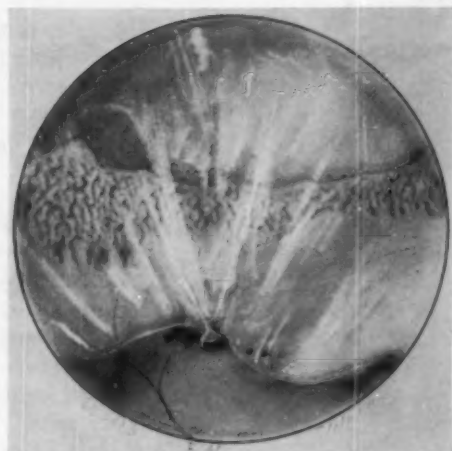


Fig. 34 (Teng and Chi) Subclinical case, Type II liquefaction. Local vitreo-retinal adhesion with pre-existing hole due to a rosette. U-shaped tear resulted.

Examination showed that the right eye was 27 by 24 by 23 mm. in size. About 13 to 14 mm. behind the limbus, between the inferior rectus and the external rectus, there was an area three to four mm. wide showing signs of coagulation. The eye was cut in a vertical plane. The vitreous showed a Type II liquefaction, up to the level of the V-shaped tear. The tear was in the middle part of the temporal side, six mm. behind the ora serrata. The upper lip of the tear was clearly adherent to the contracted vitreous. Near the tear there were 11 visible diathermy punctures. The retina was well flattened and back in position, even though the tear had not been sealed by the diathermy.

Vogt's[†] case† (figs. 37 and 38) was reported in 1937 in *Slitlamp Microscopy of the Living Eye* (English translation) and is one of the best detailed records of retinal detachment in the literature. Re-examination of the case, however, leads us to a conclusion somewhat different from Vogt's. The difference in interpretation is largely due to recent in-

creased knowledge in the field of vitreous pathology.

The patient was a 72-year-old woman with a history of myopia of 11D. Her left eye had been enucleated for reasons unknown. In the right eye she had a cortical cataract so that vitreous examination was not satisfactory. She developed retinal detachment and a round retinal hole was found. Catholysis was done on the seventh day after onset. The condition was not much improved by the operation and a second operation could not be performed due to the patient's having severe cystitis. She died 31 days after the operation, 37 days after the onset of retinal detachment.

Pathologic examination revealed a typical Type II liquefaction of vitreous. The detachment of vitreous was at the level of the hole. Vogt noted this, but felt that the contracture of vitreous might be an artefact. There was a typical round hole, which we believe to have been the result of a detached congenital rosette. This is beautifully shown in the sec-



Fig. 35 (Teng and Chi). Subclinical case, post-cataract retinal detachment. Type II liquefaction, local vitreo-retinal adhesion with pre-existing holes. Slight detachment around the hole.

† Not in the 1955 exhibit. We wish to thank the Schweizer Druck- Und Verlagshaus, Zurich, for permission to use this case.

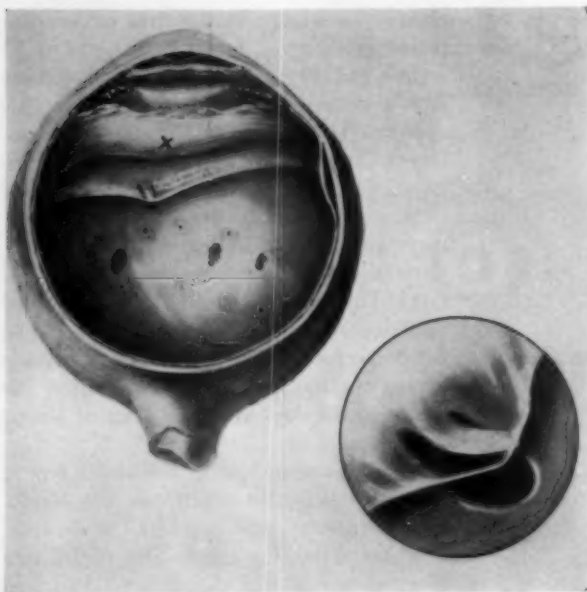
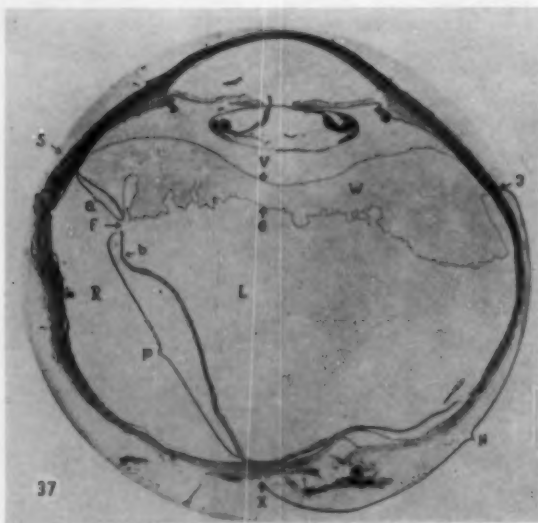


Fig. 36 (Teng and Chi). Clinical case. Type II liquefaction. V-shaped tear with local vitreo-retinal adhesion at the anterior lip of the tear. Local adhesion in continuation with senile adhesion. Postdiathermy.



Figs. 37 and 38 (Teng and Chi). From Vogt's book (figs. 2199 and 2205). A 72-year-old patient. Retinal detachment with round hole. Type II liquefaction of vitreous, local vitreo-retinal adhesion at the anterior lip of the hole and at the floater (detached rosette).

tion (fig. 38). There was a local vitreo-retinal adhesion at the point of the detached rosette (D, fig. 38) and also on the anterior lip of the hole.

DISCUSSION

Anyone reviewing the literature on this subject will find, as Duke-Elder²⁴ pointed out, that it is enormous in quantity, but it is confusing due to the fragmentary nature of the material, the lack of correlation, and the fact that many of the cases were so complicated by secondary changes that true analysis of the primary changes was impossible.

Since we have the opportunity to study such a great number of eyes in the Eye Bank Laboratory, we have been able to analyze and compare this material at the same time and in the same place. While most of our eyes are from older persons, we have also studied over 200 baby eyes. We study them first under the slitlamp, then histologically, and then try to correlate our findings with clinical cases. All cases reported here are simple cases, without complications which might obscure the true picture. This makes it easier for us to correlate and summarize our findings and thus simplify the explanation of retinal detachment.

In reviewing the various theories as to the etiology of retinal detachment, we find that it was Mueller, Leber, Gonin, and Lindner who advanced the theory of vitreous retraction, which was later summarized by Duke-Elder. He said that all authors noted vitreo-retinal adhesion, contracture of the vitreous, and liquefaction of the vitreous, but the complications of retinal detachment, such as inflammation, hemorrhage, or foreign body reaction, confused the picture.

Later Gonin proposed the theory that the primary lesion was in the retina, as in pre-equatorial choroiditis, or atrophy. Adhesion between the retina and the vitreous resulted, and the retraction of the vitreous caused the retinal tear. Many authors objected to this theory, among them Schweigger, Raehlmann, Graeff, Lauber, Vogt, Koby, and Wessely.

Hanssen examined two eyes with spontaneous retinal detachment. There was no evidence of vitreous bands, cellular membrane, or of hyperplasia of the ciliary epithelium. Nor did we observe any of these abnormalities in our cases.

In 1921, Fuchs described a number of eyes which showed little or no tendency toward detachment of the retina, although contracting retinal scars and exudate, or cyclitic membrane had distorted the retina considerably.

In previous papers we reported that the congenital retinal rosette, which was due to irregular proliferation of neuroepithelium, is one cause of local vitreo-retinal adhesion. Our present study seems to indicate that there are two essential pre-existing anatomic factors for retinal detachment: liquefaction of vitreous and vitreo-retinal adhesions. When these two conditions are present, a sudden forceful movement of the eyes or body is the precipitating factor which detaches the retina.

Until we understand better the physico-chemical properties of vitreous, we shall still be far from the solution to the problem of retinal detachment. But even though our present knowledge is limited and we cannot explain or prevent the process of liquefaction, we can watch carefully for early clinical signs of liquefaction and vitreo-retinal adhesions.

Vogt,²³ Berliner,²⁵ Schepens,²⁶ Hruby,²⁶ and others employed a slitlamp technique, some using the Hruby lens to observe the morphology of liquefaction and of posterior vitreous detachment.

Pischel²⁰ recently developed a special slitlamp technique for clinical examination of the vitreous and reported Type II liquefaction or vitreous detachment in over 90 percent of all cases of simple retinal detachment.

The presence of a vitreo-retinal adhesion, or even a pre-existing retinal hole, will not lead to retinal detachment unless there is Type II liquefaction of vitreous. Therefore,

the presence of Type II liquefaction of vitreous may be considered a first warning. Since we know that early treatment is of the utmost importance, it might be wise for patients who have this type of liquefaction to have some knowledge of the early symptoms of retinal detachment.

The second factor in the cause of retinal detachment, vitreo-retinal adhesion, is more difficult to detect. Our present study indicates that senile vitreo-retinal adhesions are almost surely present after the age of 50 years, but there is, as yet, no way of finding their extent.

The Schepens indirect ophthalmoscope, or its equivalent, is indispensable in looking for local vitreo-retinal adhesion at the periphery of the retina. The digital indentation technique for examination of the retinal periphery is especially valuable. Because local vitreo-retinal adhesions develop from congenital irregular proliferation of neuroepithelium, they are most often found in the periphery of the retina. They are in the form of rosettes or folds, which Schepens called meridian folds. Behind a retinal fold one may often find rosettes, singly or in a series (figs. 24 and 25). These may be too small and transparent to be detected by ophthalmoscopy.

Direct ophthalmoscopy should not be neglected. Because of the advantage of its high magnification, it is especially helpful in examining the region near the equator and the posterior portion of the fundus in a search for congenital rosettes or local adhesions.

Holes may seem easier to detect, but, in the case of very small holes, it may be impossible to differentiate between a cyst and a retinal hole.

When any sign of a rosette, fold, or retinal hole is found in combination with Type II liquefaction, the situation is serious.

SUGGESTIONS FOR TREATMENT

The degree of detachment of the hyaloid membrane may be used as a guide for beginning treatment. When the detachment is close to the level of the local vitreo-retinal

adhesion, it may be advisable to build a barrier all around the globe by means of diathermy. This should produce an adhesion between the hyaloid membrane, retina, choroid, and sclera to secure the detached vitreous or hyaloid membrane and prevent further detachment. We believe that the hyaloid membrane is important because it supports and protects the retina.

Any hole present should be sealed by diathermy to prevent fluid vitreous from getting behind the retina. Prophylactic operation is especially advisable if the patient is engaged in particularly active work. He should at least be told the early symptoms of retinal detachment and the importance of early treatment.

According to Reese,²⁷ cystoid degeneration of the retina can develop very rapidly after detachment. Therefore, patients who have Type II liquefaction, even in a moderate degree, plus a retinal hole or tear, should be operated upon as soon as possible.

Rest in a reclining position with binocular bandage is always a sound procedure in cases of retinal detachment. This is particularly true from an anatomic point of view because it places the point of insertion of the hyaloid membrane at the highest level. When the patient's head is in a dependent position, the hyaloid membrane and retina have a chance to settle back into their normal positions. It is a good sign if the retina settles back preoperatively.

If the retina remains separated, implantation of vitreous by Shafer's method²⁸ or a vitreous substitute is a very sound technique. This pushes back the contracted vitreous and retina and helps in the evacuation of subretinal fluid, and it is an important step in the operation. The vitreous should be injected at the central portion of the contracted vitreous (figs. 9 and 36 at X), never behind the hyaloid membrane, since this would push the hyaloid membrane forward and aggravate the situation by producing greater traction at the site of vitreo-retinal adhesion.

Diathermy to produce an adhesion between

the retina, choroid, and sclera is still the principal operation to be performed. Theoretically it should be better to build this adhesion so as to include the hyaloid membrane. A band of adhesion all the way around the globe at the level of the tear is recommended to prevent further detachment of the hyaloid membrane and retina.

In cases of retinal detachment with a thick vitreous traction band, it may be advisable to cut the band, possibly using a type of scissors developed by Thorpe. In cases complicated by numerous vitreous bands and cystoid degeneration of the retina there may be shortening or distortion of the retina, which prevents it from settling back into position. In such cases an operation which shortens the globe may be the wisest choice.

SUMMARY

In this article we have drawn distinctions between two types of vitreous liquefaction. The first type is characterized by the fact that it starts from the central core and extends to the periphery. There is no posterior detachment of the hyaloid membrane in this

type. The second type is characterized by anterior contracture of the solid portion of the vitreous, leaving the posterior portion in a liquid form. There is usually posterior detachment of the hyaloid membrane in this type.

We also distinguished between two types of vitreo-retinal adhesion: the senile peripheral adhesion and the local adhesion which forms at the site of a congenital rosette. This local type of adhesion often includes some of the immediately adjacent area.

Statistically the first type of adhesion is more prevalent. It is not usually found before the age of 30 years, but it is always found in older age groups.

Type II liquefaction of vitreous and a vitreo-retinal adhesion are invariable factors in producing retinal detachment. Any motion which produces traction at the site of the adhesion is believed to be a precipitating factor. It may cause a retinal tear and once this happens fluid vitreous may get behind the retina and separate it from its layer of pigmented epithelium.

210 East 64th Street (21).

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FUNDUS PHOTOGRAPHY BY ELECTRONIC FLASH

PART II. HIGH RESOLUTION FUNDUS PHOTOGRAPHY

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Saint Louis, Missouri

A. INTRODUCTION

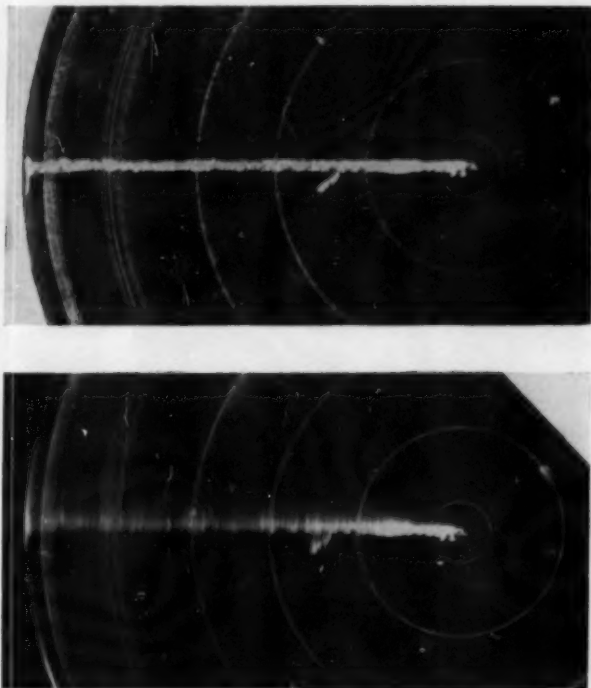
In Part I, I described the successful adaptation of electronic flash to the Bausch & Lomb Nordenson fundus camera. In this report I shall show how the same instrument can be used to take high resolution black and white photographs of the fundus.

In the past most investigators have felt that the aberrations of the human eye are of such magnitude that the resolution of the photographs made with the existing Norden-son camera could not be improved. One of

the objectives of this investigation was to test the validity of this conclusion.

With arc lamp illumination, exposures of 1/10th to 1/25th of a second are required. I felt that it was uncertain just how much of the blurring of the photograph was due to the voluntary and involuntary movements of the patient's eye. The finest movements of the fixating eye amount to two seconds of arc and occur 50 to 100 times a second.¹ With the present apparatus, the effect of even these movements can be eliminated for all practical

Fig. 1 (Drews). *Analysis of the duration of the electronic flash.* The upper photo shows part of the lathe face-plate at rest with the white radius line. The lower photo shows the same area photographed with the electronic flash while the face-plate was making 3,600 revolutions per minute.



purposes because the effective photographic duration of the electronic flash is about $1/3,000$ th of a second.*

* Determination of the effective photographic duration of the electronic flash:

Photographs of a cathode ray oscilloscope analysis of the fall in voltage across the flash capacitors during discharge allowed us to estimate an effective photographic flash duration of about $1/3,000$ th of a second, assuming that the flash effectively ceased when 90 percent of the energy in the capacitors had been dissipated.

Attempts to analyze the flash duration with the oscilloscope connected to a phototube or a sun battery failed because of the relatively slow response of these devices as compared to the very short electronic flash duration.

Direct determination of the effective photographic duration of the flash was then made. A disc of paper was mounted on a wood lathe face-plate and painted black. A white line was then drawn along a radius of the disc. The face-plate was then mounted on a "Shop Smith Mark V" and by means of the continuous speed adjustment made to revolve 3,600 rpm. This angular velocity was checked by watching the appearance of the radius when illuminated by a single fluorescent lamp: at exactly

I think that my electronic flash color photographs show an increased resolution but this of course cannot be proved. However, the efficiency of the new light source now makes it feasible to try to reduce the aberrations of the optical system, a step which would not have been at all worth while with the long exposures required with various types of arcs.

B. EXPERIMENTAL CONSIDERATIONS

The chromatic and lateral color aberrations of both the fundus camera lens system and the patient's optical system could be elimi-

3,600 rpm one sees a double fan-shaped blur which does not revolve. The revolving disc was photographed using the electronic flash, and the angular blurring of the radius on the photograph was found to be about seven degrees, even with considerable over-exposure (fig. 1).

$$\frac{7^\circ}{\frac{3,600 \text{ rpm}}{60 \text{ sec/min}} \times \frac{360^\circ/\text{rev}}{3,000}} \approx \frac{1}{3,000} \text{th of a second}$$

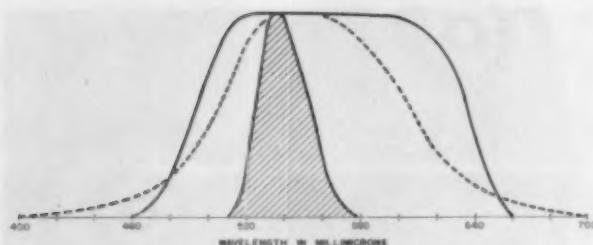


Fig. 2 (Drews). *Approximate spectral responses.*

Dotted line: the human eye (ICI Standard Observer).

Solid line: Kodak Tri-X film with a Kodak Wratten filter No. 8 (K2).

Shaded area: Kodak Tri-X film with a Kodak Wratten narrow band filter No. 74.

Ordinates arbitrary.

nated simply by using monochromatic light. After some experimentation I found that I obtained my most useful results with a Kodak Wratten narrow band filter No. 74. This filter transmits only a very narrow portion of the spectrum in the region of the mercury-green line (fig. 2). Its color gives excellent contrast between the retinal vessels and the pigment background and also shows the juxtapapillary retinal nerve bundles. The No. 74 filter absorbs about 96 percent of the light from the flash tube, but this extreme waste can be compensated by the use of Kodak Tri-X film which is rated (ASA) 20 times as fast as Kodachrome and which is capable of much higher usable film speeds.

Control photographs for comparative purposes were always made on the same roll of film on which the exposures with the narrow band filter had been taken. Thus identical film and processing conditions were assured so that differences in resolution could not be ascribed to variations in these factors. To prevent overexposure, a Kodak Neutral Density filter No. 96 of gamma 1.0 was used together with a Kodak Wratten filter No. 8 (K2). The latter gives an effective film response which is close to that of the eye (fig. 2).

All filters were mounted in simple paper jackets under the film-holder adapter, just as the 81EF color correction filter is mounted for Kodachrome photographs (see Part I, fig. 2). In this position the filters do not affect the light used to focus; otherwise this light would be so reduced in intensity that focusing would be very difficult.

All film was processed in Microdal ac-

cording to the manufacturer's recommendation.

C. EXPERIMENTS AND RESULTS

A strip of Sowerby resolution test plates was hung 20 feet in front of the Bausch & Lomb Nordenson fundus camera. Using an ordinary tungsten light source and time exposures, comparative photographs were made of this resolution test strip. With the full spectrum a resolution of about four lines per mm. was obtained; with the narrow-band spectrum a resolution of a good eight lines per mm. was obtained. Sharpness of the photographs was markedly increased by use of the narrow-band filter.

I then turned to photographing the fundus oculi. The same subject was used as had been used for my previous experimental color photographs (M. E. D.; O.S.; refractive error, $-0.25D$, cyl. ax. 165°). My criterion for excellent color fundus photographs of this subject had been the resolution of a very small blood vessel running across the lower temporal portion of the disc. This vessel was also just barely visible by ophthalmoscopy. The photographs made through the K2 and neutral-density filter combination again resolved this tiny blood vessel (fig. 3). The photographs made through the narrow-band filter in addition to being much sharper generally, resolved not only this tiny blood vessel but its even tinier branches (fig. 4).

D. SUMMARY

Fundus photographs of extraordinary resolution and sharpness have been made with the use of the Nordenson fundus

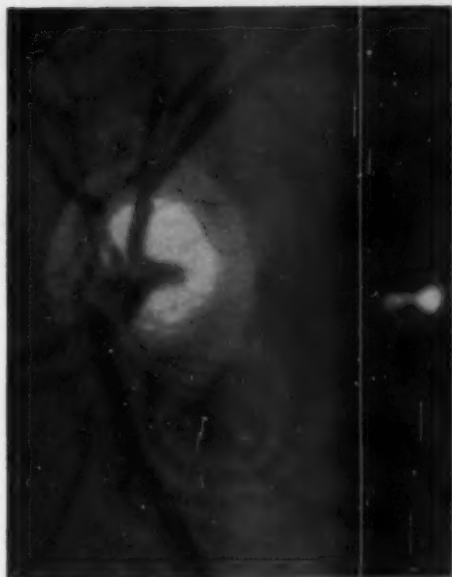


Fig. 3 (Drews). *A portion of a control fundus photograph. Kodak Tri-X film with Kodak Wratten filter No. 8 (K2). Note the tiny vessel on the lower temporal portion of the optic disc.*



Fig. 4 (Drews). *A portion of a high resolution fundus photograph. Kodak Tri-X film with Kodak Wratten narrow band filter No. 74. Note the small vessel on the lower temporal portion of the optic disc, and its branches.*

camera to which electronic flash has been adapted. Kodak Tri-X film is exposed for approximately $1/3,000$ th of a second through a Kodak Wratten narrow band filter No. 74 and developed in Kodak Microdal developer.

The method requires no special skill over color electronic flash fundus photography, the only difference being the use of black and white film and a narrow band filter.

7361 Cornell (5).

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OPHTHALMIC MINIATURE

Spectacles are among the most indispensable instruments for man. . . . If we add that spectacles laid the foundation for the invention of the microscope and telescope, whose mighty influence is powerfully exemplified in the development of most natural sciences, we shall not view these simple instruments without respect.

F. C. Donders,

Accommodation and Refraction of the Eye, 1864.

OPHTHALMOLUMINESCENCE*

DEFINITIONS AND DESCRIPTIONS OF EFFECTS

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Ophthalmology may well claim a pioneering interest in the phenomena of tissue luminescence ever since 1882 when Pflüger described the spread of weak sodium fluorescein solutions through aqueous, vitreous, and optic nerve and observed the fluorescence in white light illumination. In 1888, Straub introduced the use of fluorescein for the delineation of corneal epithelial discontinuity. Beginning in 1903, the physicist R. W. Wood devised screening filters to obtain a pure longwave ultraviolet band at 3,600-3,660 Å using carbon or mercury-arc illuminants, and modern descriptions and utility of luminescence date therefrom.

The myriad possibilities inherent in the more general employment of luminescent phenomena in ophthalmodiagnosis, operative and postoperative progress studies as described by Hague,¹ Campbell and Boyd,² and Anderson;³ in cancer detection and studies by Figge,⁴ Ronchese,⁵ Wilson;^{1b} and in screening surveys developed by Benson and Vogel,⁶ are such as to demand widespread knowledge and utility by all who work with the eye. The longwave ultraviolet 3,600-3,660 Å (Wood's light) band is not harmful for general short exposure use and produces significant optically visible tissue colors in most normal and abnormal tissues or cytoplasmic products. However, Bachem¹⁴ points out the dangers of repeated cumulative high intensity exposure of the lens to the near ultraviolet and, in view of this, it is urged that exposure to Wood's light be limited.

* Dedicated to the memory of Robert W. Wood, physicist extraordinary, whose passing on August 11, 1955, at the age of 87 years, marked the closing of an era of pioneering research. The publication of this paper was aided by a grant from the Ophthalmological Foundation, Inc., New York, Conrad Berens, M.D., Director, and from the Department of Research of the New York Association for the Blind.

Changes in luminescence from normal may be observed readily in the interpretation of a disease condition. "Fresh tissue differs from dead and decaying tissue, as also does senescent differ from juvenile tissue. Cancerous tissue may appear different from surrounding normal tissue and demarcation is facilitated," as stated in the Hanovia Company booklet.⁷ In dermatology, luminescence studies and surveys have proven to be of great value and are an integral part of thorough dermadiagnosis as discussed by Costello and Luttenburger.⁸

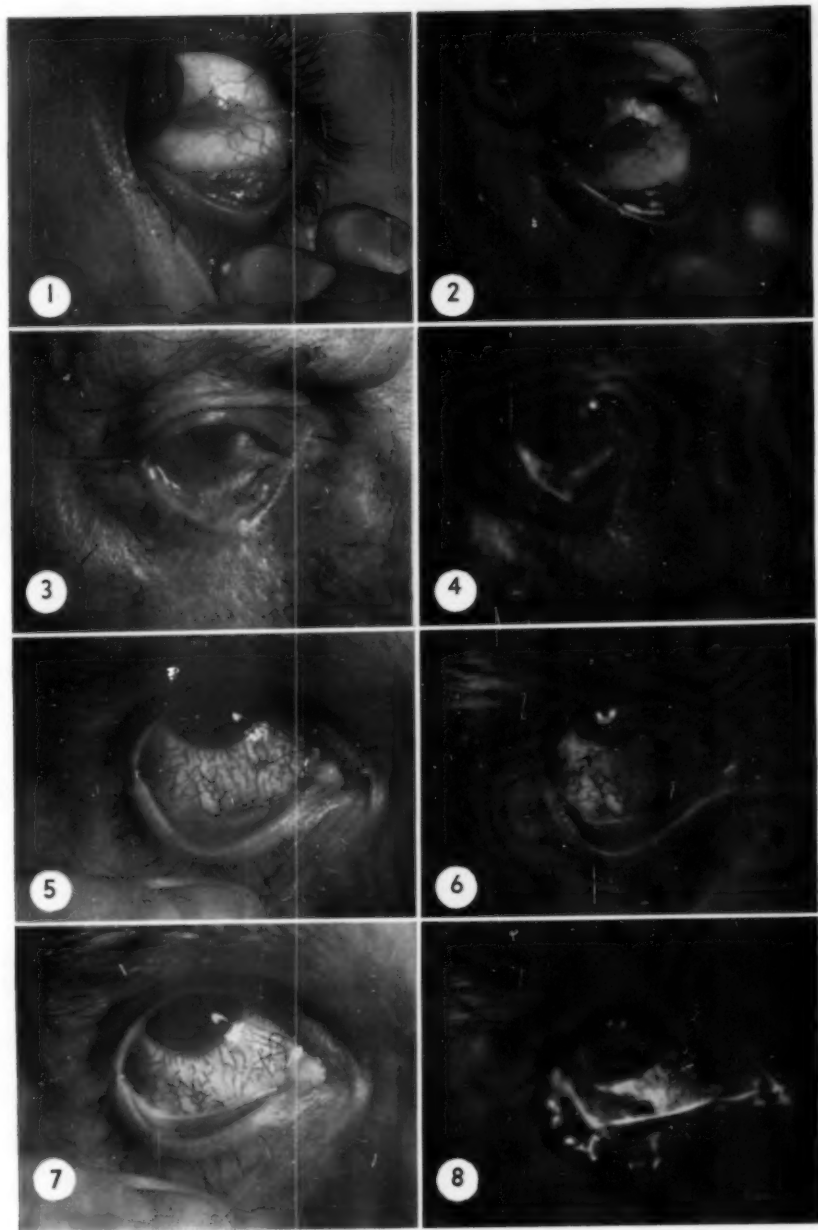
The primary or direct characteristic luminescences of the eye tissues are observable directly in the darkroom by a scopic observer employing a Wood's light source and the secondary luminescence of a fluorochrome stain is readily obtained by instillation into the palpebral sac or by parenteral administration.

THEORY OF LUMINESCENCE

The irradiation of matter by flux of light quanta or photons causes, generally, a lower-order energy emission or luminescence according to the Stark-Einstein (1908; 1905) photochemical law[†] (each molecule absorbs one quantum of radiation-inducing reaction) and Stoke's Law (1852)[‡] (emitted energy as photons does not exceed absorbed energy), the radiation emitted having a greater wavelength. Fundamental to these formalizations is Bohr's (1916) assumption that the molecule exists in certain energy states of which the lowest is E_0 , the "ground state,"

† The mathematical expression of the Stark-Einstein Law is: $E = Nh\nu = Nhc/\lambda$, wherein E is one einstein of radiation for any given λ , N is Avogadro's number, h is Planck's universal constant, c is light velocity, λ is wavelength of the radiation, ν is frequency of the radiation.

‡ Stoke's law is: $E_{\text{(emitted)}} \leq E_{\text{(absorbed)}}$ as the general case. Resonance radiation occurs where the two energies equate with perfect efficiency.



Figs. 1 to 8 (Stein). Ophthalmofluorescence.

- Fig. 1. Conjunctival melanosis. White-light flash tube; 35-mm. Ektachrome film; f:8.
 Fig. 2. Same as Figure 1 with 3,650 Å illuminance; 35-mm. Ektachrome film; f:2; exposure time 0.5 second.
 Fig. 3. Radiodermatitis and cicatricial ectropion secondary to radiotherapy of basal-cell carcinoma of right lower lid. White-light flash tube.
 Fig. 4. Same as Figure 3 with 3,650 Å illuminance.
 Fig. 5. Chemical bulbar conjunctival escharosis and erosion.
 Fig. 6. Same as Figure 5 with 3,650 Å illuminance.
 Fig. 7. Chemical bulbar conjunctival escharosis and erosion with two-percent sodium fluorescein stain. White-light flash tube.
 Fig. 8. Same as Figure 7 with 3,650 Å illuminance. This vividly depicts the marked secondary ophthalmofluorescence of uranine solutions in 3,650 Å illuminance. The uranine emits 5,600 Å in the green band, thus accounting for the typical green color.

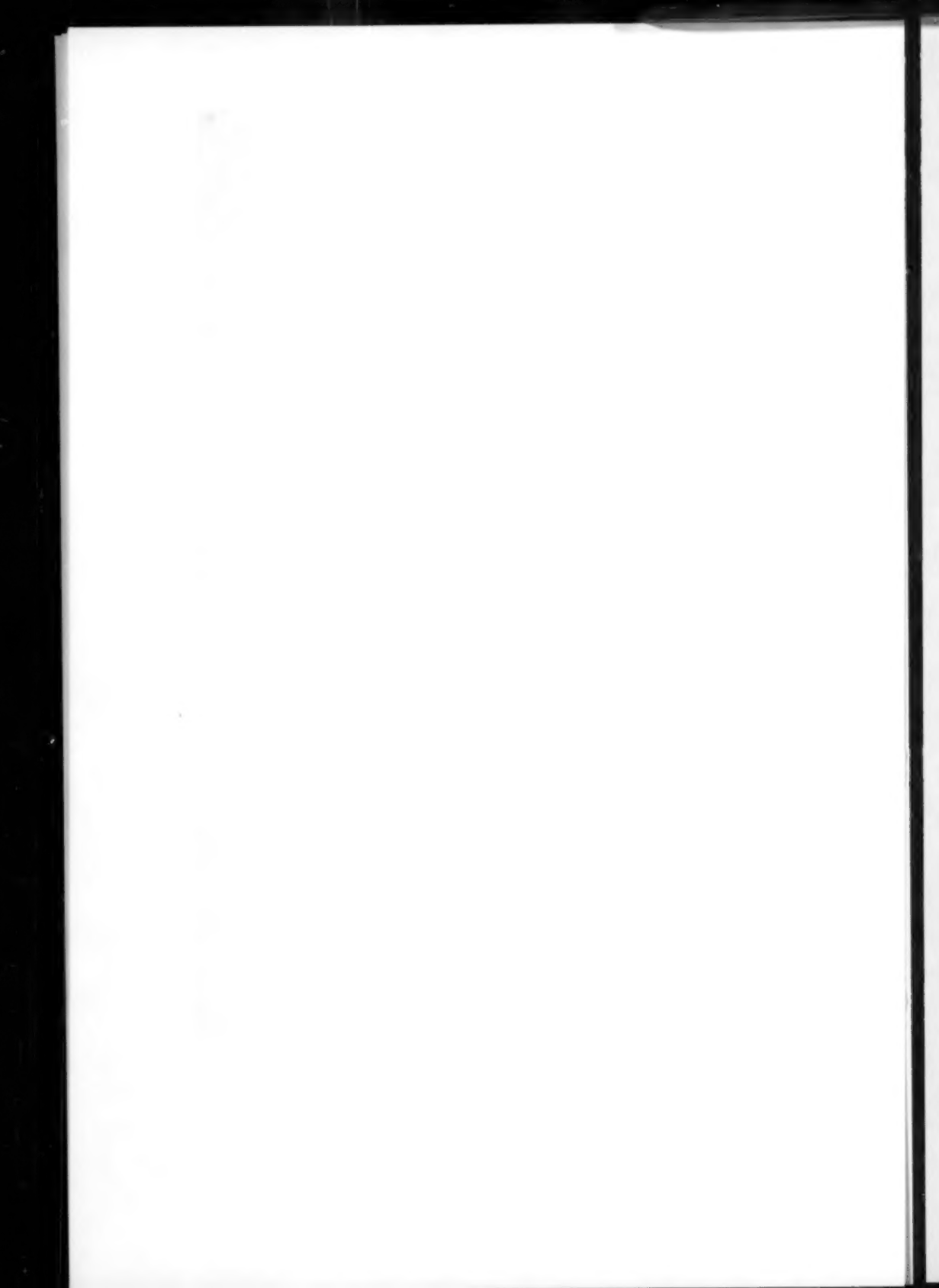


TABLE 1*
 GENERAL CLASSIFICATION OF THE EMISSIONS FROM LUMINESCENT SOLIDS

Increasing Energy of the Emitted Photon	Type of Radiant Emission	Source of Radiant Emission	Common Type of Spectra
	Gamma-ray luminescence	1. Transition of nucleons in atomic nuclei	Gamma-ray spectra
	X-ray luminescence	2. Electronic transitions in inner completed shells of atoms	X-ray line spectra
	Conventional luminescence (optical)	3. Electronic transitions in inner incompleated shells of atoms	Visible and near-visible line spectra
		4. Electronic transitions in outer (valence) shells of atoms and molecules	Visible and near-visible Band spectra
	Thermal radiation	5. Transitions of atoms and groups of atoms vibrating and rotating; also electrons as in 3 and 4	Infrared Band spectra

* From Leverenz.¹⁰ Reprinted with permission of the publisher, John Wiley & Sons, Inc., New York.

and states of higher energy E_1 , E_2 , and so forth, are called excited states. The transition of the molecule from a state of lower energy E_1 to one of higher energy E_k requires an absorbance $\Delta E = E_k - E_1$ which may be provided by a photon $h\nu_{k1} = E_k - E_1$. Upon reverse passage from E_k to E_1 , an energy emission occurs which is manifest as luminescence or "light" of the same oscillation frequency but of longer wavelength according to the relationship of $\nu_{1k} = \nu_{k1} = c/\lambda$ where c is the light-speed and λ the wavelength. Pringsheim and Vogel⁹ point out that if E_k is the result of absorbance of an optical (visible light) photon then the emission spectrum will result from electron transitions from E_k to E_0 or $E' \dots E''$ (different excited states) and, characteristically, from one single excited state the spectrum will consist of series of lines and in more complex molecules or condensed systems, sequences of broad bands will appear instead; and with further complexity, these will merge into one continuous band showing several peaks of higher intensity. Table 1 from Leverenz¹⁰ clarifies the relationships of excitation, emission, and spectral result.

Upon examining the photochemical law, certain conclusions may be derived. It is seen that E is greater as λ decreases; that is, the energy absorbed per mole increases with

short wavelength excitation and, therefore, photochemical activity is enhanced by ultraviolet radiation as compared to red light of longer wavelength. Furthermore, the selection by physicist Wood of the 3,600-3,660 Å band to perform studies in luminescence yields convenient emission of light visible to the human eye (lying between 4,000 to 8,000 Å) since the degradation of energy (according to the Stark-Einstein Stokesian, and Bohr's formalizations) leads to longer emissive wave-lengths than the shorter wave-length exciting irradiation.

The luminescent emission is governed by the spectral excitation, the atom/molecular structure, crystalline pattern, solvent, atmosphere, and temperature, as shown in other research.¹⁰ Another variable is the observing human eye, its refraction, perceptive efficiency, color vision, and state of adaptation (scotopic extent). Interpretation of tissue luminescence requires constancy and reproducibility of excitant and experience has shown that the use of mercury-arc lamps or "fluorescent" tubes provides dependable illuminant sources.

DEFINITIONS

If the excitation and emissive processes occur in times approximating the natural lifetimes of excited nonmetastable isolated

atoms (about 10^{-8} sec. for optical transitions or visible light) the process was originally termed *fluorescence*, whereas if the emissive process is of longer duration the term *phosphorescence* was applied, which embodied the prior fluorescence as a time-limited case of shorter duration. For conventional optical emissions, all persistences longer than 10^{-8} sec. are called phosphorescence to indicate an abnormal delay in return of disturbed electrons to the prior parent levels.

The term *luminescence* embraces all types of radiant emission irrespective of type or time. Ancient observation differentiated fluorescence and phosphorescence according to the 0.1 sec. persistence of human vision after the cessation of optical radiative excitation. The subjective term *fluorescence* is now ignored in the larger concept of *luminescence* because the human eye is but one of the many photosensitive devices employed to detect luminescent effects. The term is independent of the similarly derived words "luminance" and "luminosity" which pertain to the human-eye concept of brightness.

A variety of special terms has been coined which Leverenz¹⁰ describes in detail. *Chemoluminescence* and *bioluminescence* relate to chemical reactions in the inorganic, organic, and vital fields. *Fluorophors* display fluorescence only and *phosphors* only phosphorescence. The general term *luminophors* refers to all substances which convert part of the energy of absorbed photons or material particles into emitted radiation in excess of thermal radiation. Electroluminescence deals with the induction of emissive "radiative light" as a consequence of electron, beta-ray, or cathode-ray phosphor excitation as in oscilloscopes, television, and panel-lighting. This is in contrast to "fluorescent-tube" lighting which depends upon the mercury-arc spectral radiation line at 2,537 Å to excite a suitable crystalline phosphor internal tube-coating.

The bioluminescent rhodopsin-retinene process of converting optical photons into sight is part of but far afield from this discussion which is limited to effects of photons upon ophthalmotissues other than retina. As

stressed by many authors, the production of luminescence by vital tissues or protoplasmic products (skin, blood, melanin, lens, fat, and so forth) after harmful shortwave irradiation with subsequent emission of longwave photons represents a natural protective device. This is taken advantage of in the eye by virtue of the strong lenticular luminescence which converts incident ultraviolet excitation into green optical luminescence, thus helping protect the deeper retina from radiative injury. In so doing, the lens may lose its integrity and develop opacification of the denatured lens cytoplasm and cataractogenesis (Duke-Elder, volume 6, p. 6520).

Gamma-ray and X-ray radiations, as well as thermal or infrared radiations, lead to the same outcome even more intensively, owing to increased photon energy in the case of gamma-ray and X-rays and atom-molecular derangements in the case of concentrated infrared radiation.

Abiotrophic alterations are deliberately induced during radiotherapy of neoplasms or infection as a consequence of X-ray luminescence with emission of short wavelength ultraviolet photons intra- and intercellularly, and excitation of charged ions intraprotoplasmically. It should be noted that the research method of measurement of anatomic eyeball length by X-ray stimulation of the retina is an example of useful primary X-ray luminescence directly; the fluoroscope screen and intensifying phosphor-coated platens are examples of X-ray excited phosphors which then emit lower-energy photons for visual or photographic use.

It is suggested that the term *primary ophthalmoluminescence* be employed to designate those varicolored appearances of the tissues of the eye and its adnexa resulting from primary exciting illumination (paraphrased "illuminescence") with long wavelength ultraviolet light (3,600-3,660 Å, Wood's light), Hague.¹ Further, since the main fluorescein dye or fluorochrome (Pascamore and King¹¹ and Anderson⁸) in general ophthalmic use is a weak solution of sodium fluorescein (uranine) the use of the term *ophthalmofluores-*

cence should be construed as always to relate to the secondary fluorescence of ophthalmotissues stained with uranine solutions.

By thus restricting the use of the term fluorescence to its older, original definition as used in ophthalmology, future use of other dyes yielding other secondary luminescent color effects would not lead to confusion since any selectively dissolved, absorbed, or adhered fluorochrome may yield the characteristic luminescence of the dye molecule; whereas, the tissue involved may not have yielded a specific luminescence in the visible range.

If, for example, luminescence studies with iodofluorescein solutions are made, then the term "secondary ophthalmoluminescence, Rose Bengal" shall be used rather than a vague "fluorescence."

ANALYSIS BY MEANS OF LUMINESCENCE (Leverenz,¹⁰ p. 245)

"Because the luminescences of materials are determined chiefly by their compositions and structures, it is sometimes possible to identify a luminescent material by its distinctive luminescent characteristics. This analytic technique has become of practical consequence in (1) immediate identification of certain luminescent minerals, (2) identification of numerous natural and synthetic organic substances and materials, many of which exhibit distinctive changes in their luminescence characteristics when subjected to different treatments, and (3) identification of certain chemical constituents of a material, e.g., by coupling a metallic cation to an organic substance to form a metallo-organic compound having a distinctive luminescence emission."

Ophthalmoluminescence depends upon the characteristic luminescences of the normal ocular tissues, the pervading vasculature, the presence of varying amounts of pathologic deposits or alterations due to trauma, toxins, abiotrophy, or senescence, all of which affect the final color of luminescence. Neoplastic alterations because of marked cellular and vascular changes are readily remarked and

where ulceration of necrotic central portions occurs red luminescence is seen.⁴ The presence of certain metallic molecules, such as copper, may impart a greenish hue in punctate fashion as seen in arc-flash keratoconjunctivitis from copper-wire short-circuits at high voltages. In an early case this primary luminescence can be secondarily contrasted with weak aqueous mercurochrome solution to outline the punctate corneal epithelial dehiscentences as a red circle about the central foreign body as displayed with the biomicroscope.

The possibility of research to find distinctive metallo-organic luminescence is a fruitful field of cancer diagnosis. Insofar as secondary luminescence is concerned, Leverenz,¹⁰ page 250, says: "Luminescent organic dye molecules are generally characterized by conjugated double bonds where each bond in a structural formula represents two paired (shared) electrons with antiparallel spins, as in the xanthenes represented by the fluorescein molecules shown in Fig. 15. The luminescence process in these cases occurs within the closed-ring molecules, presumably by producing changes in the electron spins and distributions associated with the alternating single and double bonds. . . ."

Tables 2 to 4 itemize some of the described colors of tissues due to primary ophthalmoluminescence, secondary ophthalmofluorescence, and of foreign bodies.

Now that some tabulation and definitions of effects of primary and secondary ophthalmoluminescence has been noted, one can draw attention to certain special effects and aspects of luminescence in ophthalmology which command increasing importance.

I. THE STUDY OF MELANOSIS, LENTIGINOSIS, NEVI, AND VITILIGO OF THE LIDS

Since the lid margins are the site of conjunctival nevi, it is imperative to study suspicious lesions for pigment concentration, extent of spread, and depth of invasive vascularization, and check repeatedly for evidence of alteration or local extension (fig. 1, color photograph of conjunctival precancer-

TABLE 2
PRIMARY OPHTHALMOLUMINESCENCE
(Excitation: 3,650 Å, Wood's)

Tissue	Color
Skin, White	Violaceous variable low brightness, "ghastly"
Freckled	Covered by many lentigines hitherto invisible
Negroid	Dusky, dark. Poor reflectance
Sun-tanned	Darkly-hued but different from Negroid
Aged white	Deadish appearance
Fat	Yellowish
Muscle	Velvety-brown
Blood-serum	Yellow to olive-green
Erythrocytes	Faint violet
Crystalline lens	Aquamarine in adult Caucasians; green in immature; yellowish-green in Negroes
Cataract	Whitish aquamarine hues
Luxated lens	(Tilted) greenish-white luminescence with clear vitreous contrasted in equatorial arc (permits photoluminography)
Cornea	Clear when normal; hazy when inflamed, edematous or under pressure of glaucoma
Sclera	Slaty or whitish-bluish hue. Limbus grayish-white, pigmented. Arcus hazy
Vascularization of limbus-interpalpebral zones	Dark reddish-violaceous perilimbal areas
Pterygium	Individual arcades, loops or channels contrast vividly against scleral background
Pinguecula, Bitot's spots	Whitish-gray, foamy matrix, body, neck. Advancing epithelial head violaceous heaped-up crescentic shelving into clearer corneal edge
Meibomian lithiasis	Brilliantly whitish sudsy or foamy, streaked
Chalazia	Chalky-white granules
Melanopigment	Deep reddish-violet concentrated areas surrounded by tarsal pinkish-lavender plate
Hematoporphyrins	Dusky-brown to warm or blue-violet cold black
Icterus	Reddish to brilliant crimson, as in cancer or sebum of comedones, follicles or in lupus
Argyria	Yellowish-brown scleral hue
	Dark slaty hue in scleral tissues; deep bluish-violet in conjunctiva

ous melanosis of bulbar conjunctiva in visible light and Wood's light). The 3,600-3,650 Å light is unique in its ability to display the depth and extent of melanosis and aids greatly in planning excisions. Vitiliginous

patches luminesce brilliantly white and the surrounding skin contrasts the lesion with its darker hue. Pregnancy chloasma is more conspicuous. The blue nevus and black mole appear jet-black. Congenital hemangiomas,

TABLE 3
SECONDARY OPHTHALMOFLUORESCENCE
(Excitation: 3,650 Å, Wood's. Solution: Sodium fluorescein, two percent)

Tissue	Color
Bowman's membrane	Brilliant yellow-green; 5,000 to 6,000 Å
Corneal stroma	Very brilliant yellowish-green
Subconjunctival and episcleral connective tissue	Brightish yellow-green against tarsal or scleral background
Leaking aqueous (Seidel's test)	Streaming yellowish green from leak
Chronic or plain catgut	Tinted yellow-green, surrounded by violaceous reaction
Iris pigment in prolapse or iridencleisis	Very dark blackish-brown, green staining area at summit or in proximity, positive Seidel's test, if dehiscence conjunctiva
Eschars: thermal, radiative or chemical	Stain intensely according to depth, brilliantly yellowish green
"Murine" stain due to berberine content	Brilliant yellow on lids or cheeks (skin dye)
Double staining: Fluorescein-Mercurochrome 1% aqueous	In dendritic ulceration—green or red central matrix; blue peripheral pervasion or
Counterstain: 1% aqueous methylene blue	Red central core with mercurochrome and green fluorescein pervasion surround

TABLE 4
FOREIGN BODIES

Metallic	
Ferric-black	Darkly reflectant according to type of metal or oxidation films
Cupric-greenish	
Glass or vitrine bodies	Refractile violet or brownish if tinted with uranium salts or didymium
Ceramics	Brownly violaceous, porcelainized types reflectant, mottled
Plastics (clear)	Opalescent yellowish (according to chemical type)
Gunpowder, shotgun	Blackish
Gunpowder, firecracker	Bluish-black
Industrial chemicals: Crystals	Luminescence according to structure in great color variety (Radley and Grant)

port-wine marks, or telangiectasia, and senile angiomas appear darkish blue-violet with an overtone of the reddish vascularization.⁸ Visible lentiginos are starkly defined and many invisible taches are revealed.

II. THE STUDY OF SUPERFICIAL NONPIGMENTED NEOPLASMS

The extent of involvement of the skin in epithelioma is more readily demonstrable with Wood's light illuminance and aids surgical excision, or radiotherapy. Satellite, or daughter, lesions of melanoma may be detected. In ulcerating malignancies, or in those near the skin, blood pigment changes with concentration of hematoporphyrin yields red luminescence with 3,650 Å light. Parenteral infusion of a solution of hematoporphyrin hydrochloride (in 0.166 M. sodium lactate solution) leads to similar rendition of red luminescence by concentration in the vascular neoplasm and masses of such luminescing cancerous metastases may be demonstrated through the abdominal wall and perhaps on the iris, lids, and so forth. Figge's⁴ work in this field is conspicuous.

The long-ignored work of Clinton Wilson¹⁸ on differentiation of conjunctival tumors by Wood's light brought to notice by Lugossy's¹⁹ paper on conjunctival precancerous conditions now merits intense research (as he called for) of luminescent diagnosis of cancerous and precancerous lesions of the eye and its adnexa.

III. THE PROTEAN CORNEAL LUMINESCENT PHENOMENA

Corneal scars, vacuoles, foreign particles,

vascularization, and pigmentation are demonstrable in striking manner. In corneal edema, hydrops, or glaucoma, a greenish reluctant corneal haze is present as compared to normal clarity. This is a manifestation of stromal tissue fluid evinced as a luminescent primary relucency or haze. (Hague¹ mentions Thiel, W., 1925, as having pointed this out.) In glaucoma, when the patient has just begun to detect haloes, one can discern an early reluctant haze in many cases and, with advanced states of glaucoma, this luminescent greenish relucency is quite marked. Perhaps this provides an optical tool to screen patients with a Wood's light for ophthalmotension in mass glaucoma surveys. While so doing, the existence of pericorneal vascular injection is quickly brought into contrast. In secondary glaucoma, the demonstration is even more definite and conspicuous due to associated corneal pathology. Where uveitides occur, the deposits on the posterior corneal face may become luminescent, as well.

IV. LUMINESCENCE IN EYE SURGERY

The well-known lens capsule, cortical residua luminescence seen after extracapsular cataract extraction, or traumatic cataract as pointed out by Hague¹ are taught early in every eye resident's career. But only too frequently after only a passing mention of Seidel's test for leaking aqueous and, of course, the common, simple use of fluorescein solutions, the matter is dropped until the next resident raises his innocent countenance. Actually, it is during the postoperative phases that luminescence studies prove so helpful. Further studies of postoperative lens matter

absorption, striate keratitis and corneal edema, incisional healing by conjunctival epithelization or detection of epithelial downgrowths, descemetocoeles, iris prolapses, provide considerable information very simply at the bedside upon the first or subsequent dressings. One carries a portable battery-operated Mineralite* or extension-corded black-light fluorescent tube appliance,†darkens the room, and thus illuminance of the eye is facilitated. The observation is nontraumatic, does not cause photophobia, and requires only a loupe and some fluorescein solution to perform Seidel's test in addition. In keratoplasty, the corneal take is better studied for edema, de-epithelization, and subsequent re-epithelization, as well as peripheral leakage. With healing, the final leaving-off of the eyepad and the discontinuance of medicaments may be predicated more definitely on the disappearance of the incisional staining as the epithelium recovers the wound.

Final refraction can be confidently performed when the restoration of corneal optical integrity is demonstrable by the disappearance of the relucient luminescence caused by the postoperative corneal turgescence. In visible light, misleading (optical) transparency may lead one to expect better vision than obtains after refraction.

V. LUMINESCENCE BIOMICROSCOPY

The plethora of phenomena that are demonstrable with the binocular microscope and a concentrated Wood's light-beam aid in many diagnostic studies. Primary luminescence is conspicuous and the brilliant fluorescence with uranine is ever helpful. The outlining of the anterior tear film to aid in the delineation of the optic section is well known and a further contrast can be added

by the conjoint raying of the eye with a source of 3,650 Å placed conveniently or supported on a stand (figs. 9 and 10).

In early trauma, determination of lens luxation is facilitated by the contrast of the lens luminescence afforded by a beam of Wood's light and with the biomicroscope one can detect tilts or subluxation more readily than if one observes the transparent lens in white light. In trauma, the delineation of fluorescein or mercurochrome stained corneal abrasions, lacerations, wound-tracks, perforations, and descemetocoeles proves a valua-

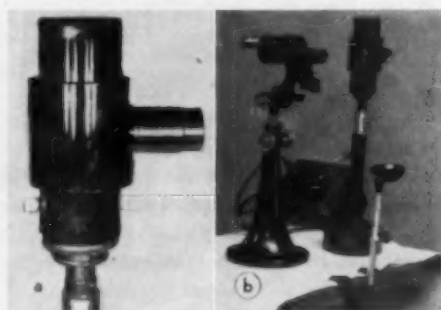


Fig. 9 (Stein). (a) The Bausch and Lomb Hildreth lamp housing with cobalt glass filter from the Bausch and Lomb Ortho-illuminator. (b) The vertically mounted Hildreth H85-C3 General Electric mercury-arc lamp in Bausch and Lomb housing, as used with their biomicroscope.

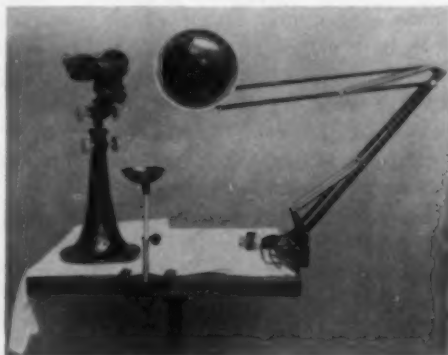


Fig. 10 (Stein). Luminescence biomicroscopy set-up, with General Electric H100-Sp4 mercury-arc lamp, showing Luxo adjustable arm and Barnet table clamp chinrest.

* Mineralite (Ultra-Violet Products Company, San Gabriel, California).

† The Burton Hand-Magnifier is most efficient and handy with two four-watt "black-light" fluorescent tubes (see fig. 13). (Burton Manufacturing Co., Santa Monica, California.)

ble way of rendering the lesions conspicuous and permitting photography with Ektachrome film. The common factor is epithelial discontinuity, disruption, or dehiscence.

Thermal, chemical, and radiative escharosis produce the same results in different ways and are vividly fluorescent with uranine solutions and thus can be photographed (color photographs 5 to 8).

Traumatic destruction of overlying conjunctival epithelium exposes subconjunctival or episcleral connective tissues which yield a brilliant uranine secondary ophthalmofluorescence. Tarsal conjunctival disruptions are also vividly stainable, especially after heat, lime, or alkali and acid burns.

In chemical injuries this staining facility proves most advantageous as in visible light merely the vascularization is seen but when stained the entire gross area of damage of globe or lid is beautifully demonstrable (see color photos). Foreign bodies of all types are rendered conspicuous by perfluorescence. Opaque particles are seen as black spots in a greenish-stained matrix; clear particles are made visible by outlining with greenish tear fluid if superficial; or refractility with purplish Wood's light if deeply situated.

Corneal ulceration of all types, bullae, vacuoles, cysts, epitheliolysis, and dystrophies, Bitôt's spots, foam cells, lipid deposits, and pinguecula are all particularly and beautifully studied after appropriate stain with uranine, mercurochrome, or Rose Bengal (di-iodo-fluorescein) and then bringing out the secondary luminescence with Wood's light illumination.

Pterygia on the perilimbal zone are well demonstrated with primary, and then, after uranine stain, with secondary luminescence, Hague.¹ The advancing pterygial head encroaching on the corneal epithelial layer displays a heaped-up mound of luminescent purplish tissue primarily; the neck and body of the pterygium displays the thickened whitish bands of connective tissue and the reddish-purple deeply vascularized stroma

are seen entering from the base. Thus an idea of prognosis and progress of the pterygium is gained which is not so readily gleaned from visible light observation.

All of this should be done with the low-power followed by the high-power objective of the binocular microscope. All lenses should be scrupulously clear and clean. Coated lenses are superior for light transmission. Luminescence biomicroscopy is invaluable in study of anterior segment and adnexal lesions suspected of malignancy. Excision should be preceded by a meticulous luminescence survey, a white light, and luminographic picture in color and then a correlation of pathology and luminescence. Thus, Wilson's¹⁸ suggestions for studies of precancerous lesions will bear fruit.

The myriad tarsal and tarsal-conjunctival diseases, meibomian cysts, lithiases, and granulomas or chalazia are significantly shown in full size and depth both in primary and secondary luminescence. The use of uranine may demonstrate additional determination of epithelial connection. The study of the lid margin—its pigmentary, inflammatory, and infestory states—is capable of extraordinary pictures.

There is no better way of displaying the full degree of conjunctival melanosis than by primary illumination. In visible light the observer sees only a meager brownish deposit in most cases and the slitlamp little aids the study. The peripheral extent is poorly delineated and aid for excision planning is poorly obtainable. But with Wood's light, the width and depth of melano-deposition may be quite surprising, especially when employing conjoint primary luminescence and secondary fluorescence (uranine) and adjacent white light slitlamp beam (see color photographs, figs. 1 and 2).

In Wood's light "photo" luminography of an opalescent cornea (wherein the pupil is hidden in white light) clear demonstration of the pupillary border and especially the barring of posterior synechia by virtue of contrast of iridopigment against the ultramarine lens



Fig. 11 (Stein). Anterior segment photoluminography set-up. Note that the flash-tube is used without reflector and end-on to minimize corneal reflection. The flash-tube is a Braun (Nürnberg, West Germany) CeBe II Permaflash.

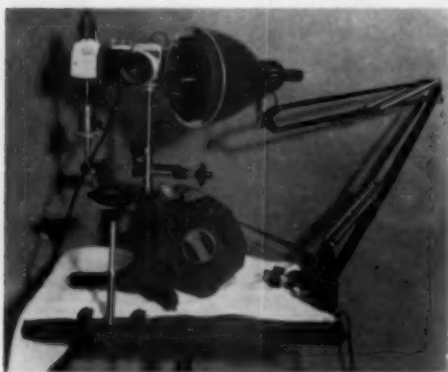


Fig. 12 (Stein). High intensity photoluminography apparatus. This is a variation of the set-up shown in Figure 11.

luminescence is obtained. Pupillary irregularity, crenation or dentation, pigmentary deposits on the anterior lens capsules and iridofenestration are delineated admirably by the lens luminescence contrast.

VI. LUMINESCENCE IN DERMATOLOGY

The dermatologic profession deserves full credit for the early and widespread use of Wood's light for diagnosis of affections of the skin and dermatologic literature is replete with many descriptions of the myriad skin diseases as seen in primary illuminance. The demonstration of fungus infections or the various tinea is a conspicuous screening achievement—*microsporon lanosum* or *microsporon audouini* luminesce a bright pale-green. Pediculi are thrown into more visible relief against the dull background of hair. In phthiriasis palpebrarum the nits at the base of the cilia are rendered a characteristic bright grayish hue and the attachment to the shaft of the cilium is discernible quickly and with a loupe the head of the nit is seen delving into the follicle. Pityriasis versicolor luminesces a dull-yellow. Syphilology employs luminescence to demonstrate fading secondary syphilitic eruptions and forming maculopapular lesions. Clinically healed moist papules and mucous patches can still be seen as dusky pinkish-red lesions with an opalescent sheen. Syphilitic papules appear as dusky-blue with a grayish tone surrounded by a pale halo of devascularization.

The chronic dermatoses are especially well studied as to course, remissions, and cure. Many symptomatic advanced lesions invisible to the naked eye may display characteristic luminescence with Wood's light. Lupus erythematosus shows powder-blue discolorations even during remissions. Evolving lesions of erythema induratum and the scars left by these lesions are distinctly visible as dark-blue luminescence. Verruca vulgaris has a silvery-white luminescence as do many other keratinizing lesions and differentiation from molluscum contagiosum is facilitated by the fact that molluscum is not luminescent except for the speck of keratin surmounting the lesions.⁸ The dysvitaminoses of the skin are also diagnosable with the aid of Wood's light. Eczemas, psoriasis, seborrheas, kera-

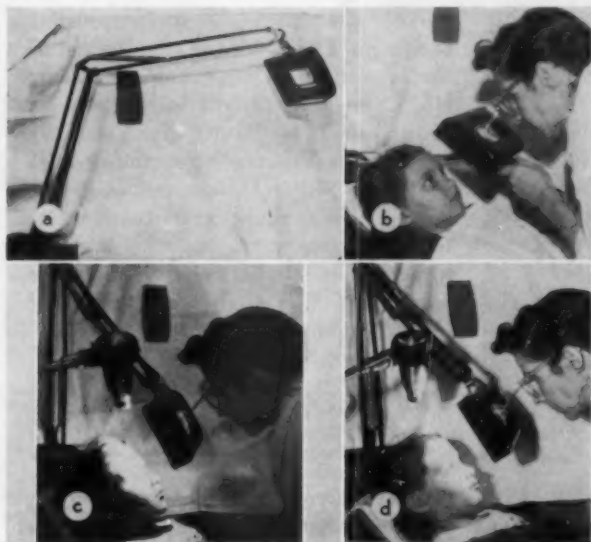


Fig. 13 (Stein). Observation through the large convex lens of the Burton unit with its binocular loupe increases magnification and stereopsis. (a) The Burton Fluoro-Magnifier. (b) Use of hand unit of Burton lamp and binocular loupe. (c) Combined flux from black-light tubes and white-light Ortho-illuminator. (d) Black-light flux from Burton unit only.

otic changes, precancerous lesions, cicatrices and keloids, radiodermatitis (see color photographs, figs. 3 and 4), skin test persistent stains, from serodiagnosis as well as from medicament, poisons, dyes, or ointment bases are some of the topics described in the dermatologic literature.

VII. INSTRUMENTARIUM

The ophthalmologist is fortunate in having many devices available commercially for the production of the 3,600-3,660 Å Wood's light band. These are usually operated with the common 110 volt AC, but battery-operated field models are also made for use in mineralogic prospecting, criminology, and food inspection. Herbach and Rademan, Inc., 1204 Arch Street, Philadelphia 7, Pennsylvania, can supply every type of black-light mercury-arc or fluorescent lamps. This may be as fully manufactured items or in kits to construct the apparatus one's self.

The "fluorescent" black-light tubular lamps contain a special phosfor whose radiation peaks around 3,650 Å and are constructed of a special (Nico) glass containing nickel

and cobalt oxides that act as an integral filter. Thus one can set up wall or ceiling units over the examining chair (fig. 14) to provide a flux of illuminescence for screening work.

Smaller banks of four, six, or 15 watt units may be used in hand illuminescors, as in the Burton Magnifier Light* which uses the four-watt tubes or the "Blak-ray" models manufactured by the Ultra-Violet Products Co., Inc. This company makes the Mineralite and a large heavy inspection portable lamp which is most durable and suitable for photography. It also supplies battery lamps for ambulant use.

The Hague "cataract" lamp of the American Optical Company is known to ophthalmologists. Many dermatologists employ the Hanovia Company's "Fluorolamp," which is similar to the Hague lamp in design and construction. All these latter, heavier models employ the General Electric

* The adjustable support of the Luxo Lamp Company, Tuckahoe, New York, lends itself beautifully for mounting the Burton Fluoro-Magnifier lamp and the H100-SP4 mercury-arc lamp and Corning Glass filter roundel supplied by Ultra-violet Products, Inc. (figs. 10, 12, 13, and 14).



Fig. 14 (Stein). Examination chair set-up with Burton Fluoro-Magnifier and Luxo adjustable lamp support. The placement of the Bausch and Lomb Ortho-illuminator facilitates alternation of white-light and Wood's near ultraviolet light during the examination.

Company 100-watt H100-SP4 mercury-arc internally silvered bulbs. A special NiCo glass heat-resistant Corning Glass filter roundel to limit the range of the emission to 3,600-3,650 Å is required for screening.* The rated lumens are 2,300, the life 1,000 operating hours.

The H100-B4 is the same arc-type jacketed with an integral filter glass and does not require screening. However, its lack of internal silvering with paraboloid shape that characterizes the SP4 prevents concentration of the radiant flux. It is useful for chair diagnosis, contact-lens work, and surgery.

Biomicroscopy requires the intense beam of the H100-SP4 or H85-C3 General Electric mercury-arc lamp.

It should be pointed out that special starting ballasts and transformers, larger Admed sockets, and heavy wiring are needed for these lamps, whereas the fluorescent tubes

can be interchanged in ordinary lighting equipment. I have devised (fig. 9-a and b) a simple stand for supporting the Hildreth H85-C3 General Electric mercury-arc lamp housing made by Bausch and Lomb Company for its binocular ophthalmoscope as a source of red-free illumination for funduscopy. This stand permits conjoint or alternate raying of the eye with Wood's or white light and facilitates biomicroscopy. The cobalt glass filter of the Bausch and Lomb Optical Company Ortho-illuminator may be utilized for filtration.

VIII. COLOR PHOTOGRAPHY OF PRIMARY AND SECONDARY LUMINESCENCE (LUMINOGRAPHY)

I have recently succeeded in making rapid exposure color photographs of luminescent phenomena. A reflex-type camera such as the Praktikon or Exakta, with a 58-mm. Biotar, f:2 coated lens, a 6.0-cm. extension tube, and 35-mm. Ektachrome film (Eastman Kodak Company) were employed. Another setup is the addition of the 13.5-diopter Keeler Optical Company Achromat as a front-element magnifier. This is held by a Tiffen adapter-ring to the front-cell of the photo lens and requires a one cm. extension ring to provide large images of the subject eye (fig. 15). Exposure time for this set-up is one-half second.

Illuminescence is obtained with the SP4 General Electric mercury-arc lamp which concentrates an intense beam of radiation



Fig. 15 (Stein). The Praktikon camera with extension tubes and +13.5 achromatic magnifying front-lens (Keeler Optical Co.) with Tiffen retaining ring on Biotar f:2, 58-mm. lens.

* Roundel, obtainable from Ultra-Violet Products, Inc., San Gabriel, California (figs. 10 and 12).

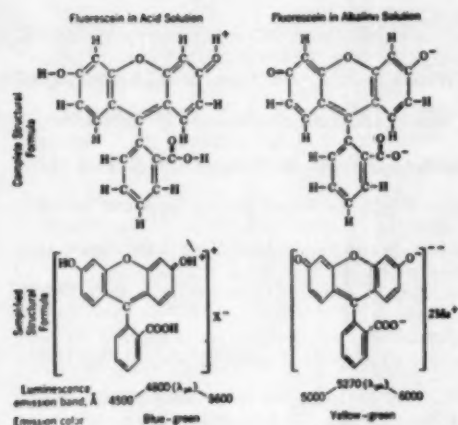


Fig. 16 (Stein). Structural formula and emission bands of fluorescein. (From Leverenz,³⁸ Figure 64. Reprinted with permission of the publisher, John Wiley & Sons, Inc., New York.)

screened with a Corning NiCo glass filter to pass the 3,600-3,660 Å band. This lamp is held about three inches away from the eye being photographed, Ronchese,⁵ Benedek,¹² Figue and Clarke.⁴³

The transparencies are developed and mounted by some suitable commercial developing house, but kits are available from the Eastman Kodak Company for one's own darkroom work. Similar film and kits are likewise obtainable from the Ansco Company. Reproductions and enlargements are inexpensively procured and make beautiful studies. It is hoped that the foregoing will lead to extensive descriptive work by ophthalmologists so that our knowledge will broaden as we gainfully employ the "black-light" discovered by physicist Wood in 1903. The widespread employment of luminescence in criminology, mineralogy, gemology, fluorophotometry, "fluorescent" lighting, fabrics, engraving, stage lighting, theater design, biochemistry, fluorometric analysis, paints, plastics, dyes, chalks, printing-inks, petroleum

research, inorganic chemistry, organic analysis, in biologic studies, educational demonstrations, food science and textiles, speaks well of the utility of luminescent phenomena (Radley and Grant).

SUMMARY

The applications of luminescence with longwave ultraviolet light filtered by Wood's filter to the narrow 3,600-3,650 Å band are discussed, defined, and described with relation to ophthalmology. Certain special aspects with reference to studies of melanosis, corneal luminescent phenomena, postoperative appearances, and utility in biomicroscopy are stressed. Photography with Ektachrome film is described and illustrated. The available instrumentarium is discussed to aid procurement of a variety of apparatus fully manufactured or for self-build kits. It is desired that this effort contribute to further ophthalmologic elucidation of a most useful diagnostic and research tool already under intense employment in many other related sciences.

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A METHOD OF MAKING AND IMPLANTING MINUTE PELLETS OF SOLID SUBSTANCES*

AND SOME OF THEIR IN VIVO USES

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The use of carcinogens for local action is not new.¹ Subcutaneous locations and large organs as brain, lung, uterus, liver, and so forth in adult and young animals^{2,5} have been favorite structures of attack because of accessibility and ease of delivery into such roomy sites.

The problem of carcinogenesis in embryonic structures has been limited to embryonic tissue transplanted into the anterior chamber of the eye or intramuscularly in adult animals.³ The use of transplanted tissue has several obvious disadvantages: the reaction of the host to the presence of the embryonic tissue, host reactions to the carcinogen itself, and the fact that the tissue to be studied has been removed from the embryonic, metabolic, and hormonal environment. The difficulty attendant upon the implantation of carcinogens

* From the Department of Research, Wills Eye Hospital, Irving H. Leopold, M.D., director. This work was supported by a grant (#254A[T]) from the Damon Runyon Fund, Inc. We wish to thank Miss Anna Bilyi for her interest in the photographic work.

into the fetus in utero are the possibility of the death of the fetus, abortion, and the death of the mother.⁴

The consistency of embryonic tissue itself prevents manipulation, and the trauma of handling the uterus and the surgery involved are all obstacles which have prevented heretofore any intensive studies along this investigative line. This laboratory was stimulated by an attempt to start retinoblastoma via the embryo and newborn animal eye. The problems of a method of manufacture of minute pellets of carcinogens of known weight and a means of implanting them into the embryo naturally presented themselves. A means of accomplishing this and one which is adaptable to other delicate structures has been developed and may be of general interest to those working on carcinogenesis, or other problems presenting a similar requirement.

The creation of pellets of minute size and of such firmness that implantation is practical is based on the observation that passage of a needle of a given diameter a given number of times through a finely powdered substance will cause such packing, of the substance to be studied, into the barrel that a firm cylindrical pellet is formed. This can be extruded by a close fitting stylet. After the pellet is made the same needle is used for inoculation purposes. The most satisfactory container is, as in Figure 1, formed of a small test tube, 5.0-cm. long by 5.0 mm. in diameter, with a soft rubber plug filling as much of the lumen as is necessary for different needle lengths. This cushion functions both to prevent blunting of the needle and to facilitate packing of the powdered substance by means of its resiliency in its small but definite bulging into the lumen of the needle which with each thrust forces a certain amount of powder into the barrel.

The needle is thrust between the wall of the test tube and the soft rubber plug with bevel turned toward the latter. This action pushes and compresses the carcinogen into the barrel via the responding elasticity of the rubber. The layer of powdered substance over the

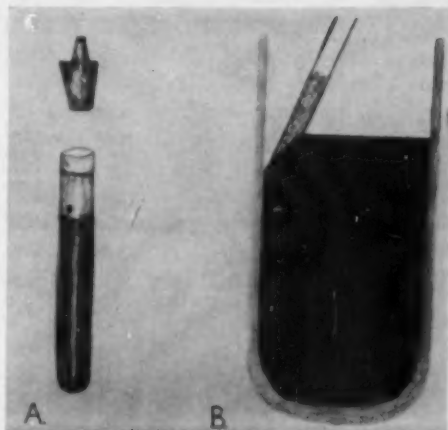


Fig. 1 (Kaczurowski and Vogel). Rubber plug in bottom of tube pushes powdered substance into needle with each thrust.

rubber should be at least 1.0 cm in depth. A smaller quantity of compound in the container can cause a diminution in pellet weight. An oblique needle bevel may occasionally tend to break the pellets on extrusion.

Figure 2 represents changes in weight of the pellets following different passages. It is seen that the more important increase of weight is in the 10-to-20 passage group, and slight thereafter. This indicates that as far as weight is concerned it would be sufficient to limit the number of passages to 10. The pellets, however, are soft and breakable.

Figure 3 shows the correlation between the hardness of the pellets and the number of passages. After 20 passages, the increase in weight is not large and the consistency of the pellet is soft. After 30 passages, there is a slight further increase in weight which does not appreciably progress with the next series.

These pellets are hard and well formed and are best for our purposes. The weights given for needles No. 17 to No. 25 are the average found for 10 pellets, each made in the same manner. For needles No. 26 and 27 the average was that of 50 individual pellets each made in the same manner. Because the making of such minute objects depends partly on manual dexterity it is probable that each

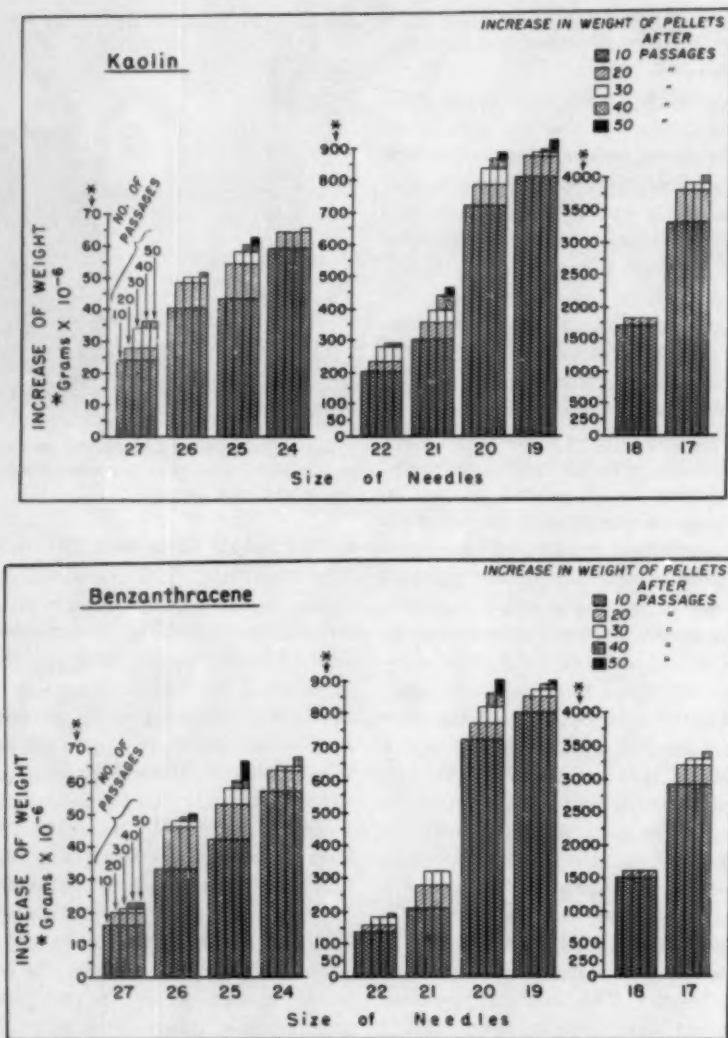


Fig. 2 (Kaczurowski and Vogel). Increase in weight with number of needle passages through powdered substance of kaolin and benzanthracene.

laboratory using this method will find it necessary to standardize its own pellets.

IMPLANTATION OF CHICK EMBRYO

The extreme mobility of this gossamerlike embryo as it sits on the yolk sac can be likened to that of a delicate object perched on a mobile ball-bearing. The slightest touch causes it to reel away from the object push-

ing it and if one attempts to hold it by means of forceps or by the allantoic membrane, he soon finds that he is grasping only a crushed and mangled remnant. After trial and error, the approach found most satisfactory for accessibility of the eye was through the air sac. Four steps of manipulation with the chick embryo are presented in Figure 4.

A sterilized carborundum disc is used in

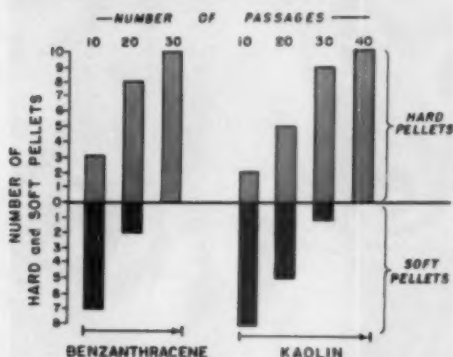
CORRELATION BETWEEN HARDNESS OF PELLETS
AND NUMBER OF PASSAGES

Fig. 3 (Kaczurowski and Vogel). The majority of pellets formed by 10 needle strokes are soft; 30 strokes form hard pellets.

making an opening 1.0 by 1.5 cm. and the window thus formed is easily moved with a special hook knife made from a razor blade. After this part of the shell has been removed, the inner shell membrane is split or partially retracted carefully so as not to damage the

amnion and vitelline membrane which contains blood vessels. Rupture of these large vessels causes hemorrhage and usually death of the embryo.⁶ After exposing the embryo the eye is implanted (infra) with a pellet and the opening in the shell is sealed by ceresine wax and a flamed cover slip.^{6a}

The instrument* for this implantation overcomes the above-mentioned manipulative difficulties of this and other fragile tissues by taking advantage of their momentary inertia, Figure 5.* The essential moving part (a) is borrowed from the Frank-lancet and is the sudden mechanical force which drives a perforating needle (e) into the embryonic site selected for implantation. A second spring (c) released by deeper pressure on button (b) immediately drives a closely fitting stylet through the perforating needle and extrudes the previously packed pellet into the organ selected for study. The best size needle for this work on the chick is the

* The tooling of this instrument was done by F. J. Todd, 201 Forrest Avenue, Narberth, Pennsylvania, and they are available through him.

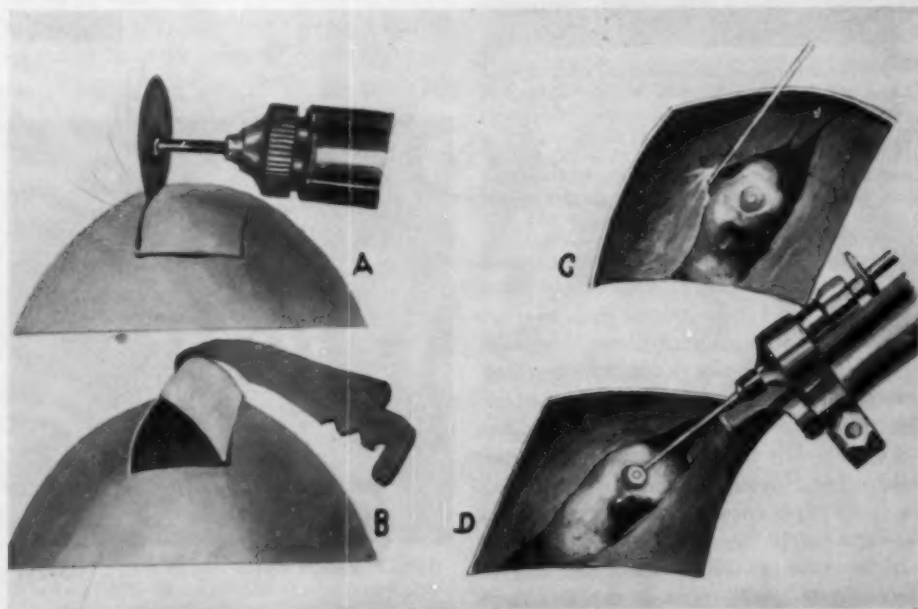


Fig. 4 (Kaczurowski and Vogel). Procedure of opening of air sac and retraction of membrane to expose chick embryo.

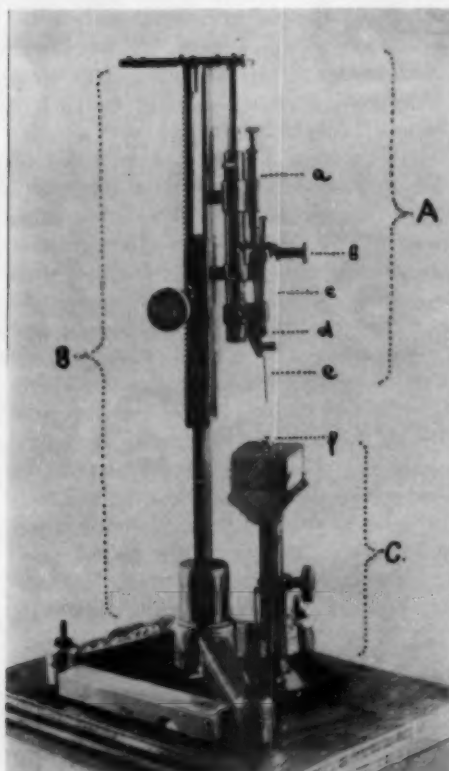


Fig. 5 (Kaczurowski and Vogel). Pellet injector (A) is mounted on adjustable stand (B) and is removable. Stand (C) is adjustable table with centrally mounted flashlight for transilluminating pregnant albino mice and newborn mice. (a) Frank-lancet which drives perforating needle. (b) Button: initial pressure releases perforating needle; deeper pressure releases the spring driving stylet. (c) Stylet spring. (d) Guide flange for needle hub. (e) Perforating needle. (f) Transilluminating light.

No. 27. The percentage of accurate implantation into the posterior chamber of the chick embryo eye has been possible in 70 percent plus of operated embryos.

Figure 6 shows the needle approaching the embryo eye. Increased difficulty in striking the eye is experienced as the embryo reached the eight- to 10-day stage because of rapid fetal movements. With practice skill can be acquired which will overcome this to a large extent. The best stage for implantation of



Fig. 6 (Kaczurowski and Vogel). Loaded needle approaching chick embryo eye after exposure through shell.

pellets into the eye is in the five- to seven-day-old embryo. Of the embryos subjected to this technique 60 percent survived to hatching.

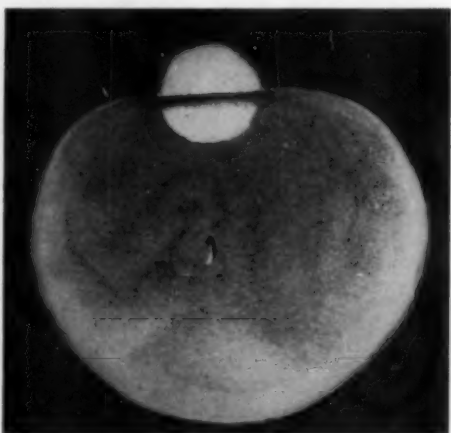


Fig. 7 (Kaczurowski and Vogel) Pellet implanted at the eight-day stage of development of the chick embryo, suspended in the vitreous of the newly hatched chick.

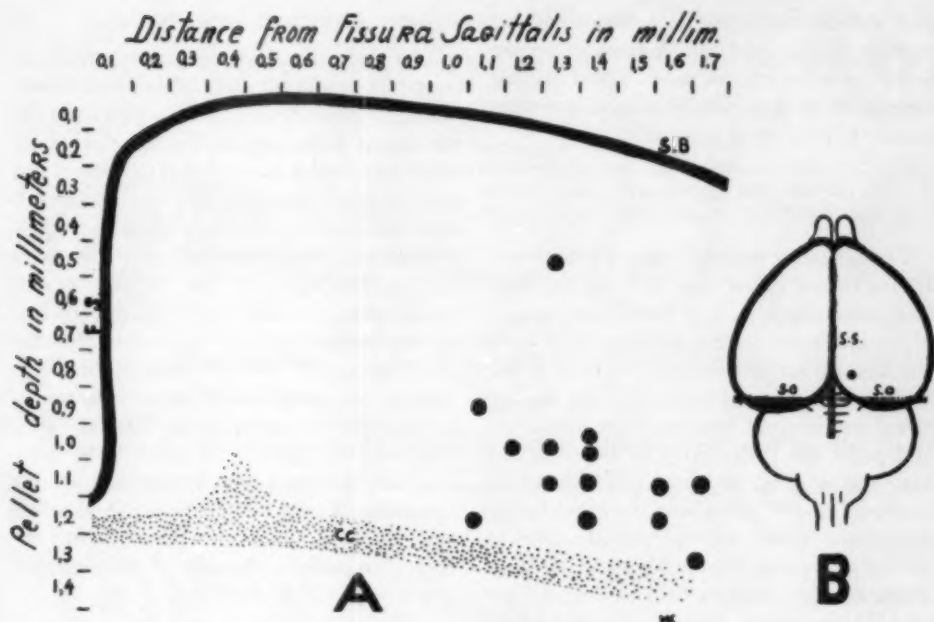


Fig. 8 (Kaczurowski and Vogel). (A) Location of pellets implanted intracerebrally in 14 adult mice. (B) Surface landmark found by dropping horizontal and vertical lines from first point of formation of sagittal and occipital sutures. (FS) Fissura sagittalis. (SB) Surface of brain. (CC) Corpus callosum. (SO) Suture occipitalis. (SS) Sutura sagittalis (interparietalis).

Figure 7 shows a pellet of dibenzanthracene injected at the eight-day stage still suspended in the vitreous of the eye of a newly hatched chick. The brain of the chick which is a larger structure is easily and more readily accessible by this procedure.

MOUSE BRAIN

A. THE FETUS

Since the retina arises from the same anlage as the brain, a preliminary experiment of introducing a carcinogen (3-methylcholanthrene) into this tissue and comparing the carcinogenicity with implantations into the brain of the newborn and adult mouse* are underway and will be presented later.

The gestation period of the mouse is 19 days. Identification of embryonic landmarks

is more difficult and complications increase in frequency the earlier in the fetal development that implantation attempts are made. A mid-line incision about 3.0 cm. in length is sufficient exposure for both mobile uterine horns. The mouse peritoneal cavity is highly resistant to infection and a clean technique gives negligible postoperative infection. Ether anesthesia seems to give lower incidence of abortions.⁷ The uveal pigment is laid down in the mouse at the 11-day stage* and this spot aids greatly in identifying head landmarks at the 11- to 13-day stage. By the 14-day stage the uterine wall has thinned and the body outlines can be seen with strong focal illumination.

Pellets of 3-methylcholanthrene have been implanted into the brain of the 14-day-old fetus and these have been carried successfully to term. Autopsy has shown the majority of pellets to be located in the thalamic re-

* Swiss-Webster, F₁. Bark-Bridge Farms, Wenonah, New Jersey.

gion and the site of needle entrance midway between the eye and ear. A trace of trypan blue is included in each pellet. Those animals born with a trace of blue stain are thus known to have been injected.

B. BRAIN OF THE NEWBORN AND ADULT MOUSE

The newborn mouse is also a fragile and friable object whose skin is, however, relatively resistant even to a No. 27 needle. Use of the implanter permits avoidance of injurious holding and penetration pressure during brain implantation. Figure 8 shows the external and internal location of the needle site and pellet site respectively in 14 cases. We have successfully implanted 220 12-hour-old newborn mouse brains with 3-methylcholanthrene and kaolin without mortality due to the initial trauma. No anesthesia is used and as the kits are manually held on the platform (fig. 5) the needle guard is lowered to the injection site selected. The same procedure can be followed on anesthetized adult mice. A midline scalp incision down to the periosteum reveals the suture-line landmarks. There was no mortality from a group of 110 adult mice so injected.

PITUITARY AND ADRENAL

The endocrine and neoplastic effects of the pituitary and adrenal gland upon the various ophthalmic structures are well known. In the mouse these organs are the size of an apple seed, and it was felt that the pellets and pellet injector were well adapted to attempted local carcinogenic action. Pellets of 3-methylcholanthrene have been implanted into these organs. The approach⁹ via the base of the skull and injection through the synchondrosis occipito sphenoidalis were used for the pituitary. Of 22 animals used for development of the technique 21 showed carbon or kaolin pellet remnants in the pituitary (left lobe) and one showed the pellet in the pons. The adrenal gland was approached via the conventional flank kidney incision and pellets have been implanted in 60 mice in either the cortex or medulla. Results of these experiments will be published later.

SUMMARY

A method of making minute pellets of powdered substances and the versatility of their use in experimental pathology have been briefly described.

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PLOTTING THE BLINDSPOT

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Mariotte's physiologic blindspot may be defined as a nonseeing area in the field of vision corresponding to the position and extent of the optic nervehead, an area which has no percipient elements (rods and cones). It is a vertical oval approximately 7.5 degrees by 5.5 degrees, with its center about 15.5 degrees temporal to the point of fixation and about 1.5 degrees below the horizontal meridian. The average size of the blindspot on the tangent screen at one meter is 130 mm. by 90 mm., with its upper margin 40 mm. above and its lower margin 90 mm. below the horizontal line. Its nasal margin measures about 230 mm. from the fixation point.

There are several factors affecting the size of the blindspot. First of all, it should be noted that the size of the blindspot, as well as its distance from the fixation point, varies directly as the distance of the eye from the tangent screen; that is, its area and its dis-

tance from the center of the screen increase as the distance between the eye and the fixation point increases.

Its borders representing the peripapillary rods and cones stimulated by rays of light, the blindspot may be appreciably influenced by refractive errors of six diopters or more. Here, the changes in its size are brought about by changes in the size of the retinal images and of the blur circles. In hyperopia, the distance between the blindspot and the fixation point is greater than in emmetropia. Conversely, it is less in myopia. Theoretically, the blindspot of the aphakic eye should be 1.35 times greater than that of the normal eye. It has been shown, however, that its size, as well as its position, is within normal limits in aphakia, when adjustments are made in accordance with the refractive power of the aphakic eye.

Another factor that influences the size of the blindspot is the intensity of illumination.

In very bright visual field rooms, excitation of the entrance zone of the visual nerve is extinguished by contrast. Hence, the blind-spot tends to be larger than with relatively weak artificial illumination with the eye fairly dark adapted whereby the contrast function is probably reduced.

Reduction of the oxygen supply to the visual apparatus results in increase in the size of the blindspot. Experimental generalized anoxia may be produced by having the subject breathe an oxygen-poor mixture or by placing him in a depression chamber. Anoxia localized to the retina and optic nerve may be produced by exerting with a dynamometer a graded amount of compression, proportional to the arterial pressure of the retina, on the eyeball. Some provocative tests are based on this principle of anoxia, the tolerance to which is less in glaucoma or glaucoma-inclined patients than in normal ones.

Enlargement of the blindspot is an important sign in certain eye conditions. The enlargement may be uniform as in papilledema (from displacement of or pressure on the retinal elements around the nervehead or from the overhanging of the swollen disc over the adjacent retina), in glaucoma (glaucomatous halo), in myopia (from atrophy of the choroid), in hysteria (psychogenic), and in senility (senile halo). Or it may be localized as extensions usually on the lower or upper poles or both (glaucoma), or on the midnasal border (toxic amblyopia). Other causes of localized enlargements are occlusion of the superior or inferior branch of the central retinal artery, juxtapapillary choroiditis, and hyaline masses at the nervehead. The barring of the blindspot in early glaucoma may be included here.

Certain congenital conditions may give rise to localized enlargement of the blindspot, like some cases of medullated nerve fibers, congenital coloboma of the optic disc, undue pigment heaping at the disc margin, Bergmeister's papilla remnants, tilting of the

optic disc, and any other peripapillary aberrations.

A word must be said about the one-degree zone of amblyopia for 0.5/1,000 white test objects surrounding the blindspot. This is due to the retina's terminating gradually at the edge of the optic disc. Therefore, measurements beyond 7.5 degrees by 9.5 degrees with the 0.5/1,000 test object in the absence of any other visible peripapillary changes in the fundus should be considered pathologic enlargement until proved otherwise. According to Chamlin and Davidoff, the one-degree zone of amblyopia around the blindspot can be shown even with a 1/1,000 white test object.

Another important observation that should be mentioned is the variability of the amount of blindspot enlargement in papilledema from any cause (intracranial tumor, nephritis, meningitis, and so forth). In some cases the enlargement may be more significant than the degree of papilledema as seen with an ophthalmoscope; in others, the opposite may be true. It is my observation that nearly all authenticated papilledema cases show distinct enlargement of the blindspot. In those in which the increase in area is small, it might be conjectured that before the onset of papilledema the blindspot in that individual might have been in the lower limits of normal. This can be proved if the other nervehead appears normal. Space will not permit a complete discussion of the various anatomic and physiologic features that account for the variability of nervehead swelling in different individuals.

Because of the fact that evaluation of the changes found in the blindspot helps in the early diagnosis of glaucoma and of papilledema, it is extremely important that when these conditions are suspected the blindspot should be mapped out routinely and as accurately as possible. Equally important is the correct, proportional, and uniform recording of the blindspot finding, even if it is normal, as this will be important for future refer-

ence. I suggest that on the chart the area of enlargement or extension be cross-hatched lightly, leaving the original outline of the normal blindspot unshaded. This will allow quick contrast and appraisal of the amount of enlargement, as well as easy comparison with that of the other eye.

After the presence of papilledema, of glaucoma, or of papillitis (as from sinusitis, diabetes, and so forth) has been established, plotting the blindspot at regular intervals is of value in appraising the patient's progress. The procedure is also of great help in differentiating true neuritis (papilledema and papillitis) from pseudoneuritis. In the latter, which is associated with high hyperopia, there is no enlargement of the blindspot and contraction of the peripheral field of vision is not expected.

The blindspot, indeed, should be routinely plotted before the periphery of the fields are delineated. Strong exponents of this are authors like Berens, Duke-Elder, Harrington, Rucker, Tassman, Traquair, and Zuckerman. The reasons offered are:

First, if it is demonstrated to the patient that a blindspot exists, his interest is aroused and his co-operation is secured more readily.

Second, the patient is mentally prepared to be on the lookout for similar scotomas. In Beren's words, "He is enabled to appreciate what is required of him."

Third, the importance of fixation is stressed to him, as failure to fixate properly would doubtless result in difficulty of outlining the blindspot. In other words, if the blindspot cannot be properly mapped out, the chances of obtaining a correct picture of the peripheral fields are slim.

Fourth, if the periphery of the fields is mapped out first and is found normal, the perimetrist is tempted to skip out plotting the blindspot, and thus miss an important tool in diagnosis.

Fifth, a number of patients fatigue easily. According to Ferree and Rand, this is not due to retinal fatigue. It is my opinion that

this is mental fatigue—fatigue of co-operation, if the term is permissible. Hence, if the blindspot is plotted last, the result may not be as accurate as when done first.

Currently, there seem to be two schools of thought as to the technique of plotting the blindspot; namely, centripetal and centrifugal. In the latter method, which is more favored, the test object is moved from the non-seeing to the seeing. A third school seems to combine both; that is, the test object is moved from the seeing, through the blind area, and then out to the seeing again.

Regarding the size of test objects, most authors favor small-sized ones, rarely 20 to 40 mm. A flat round white disc is preferred as this facilitates more precise placing of the markers (black headed pins usually).

Before considering the author's technique, certain factors have to be borne in mind. First, when the test object is started from the nonseeing to the seeing area, the blindspot tends to be larger than normal. The reverse holds true with the other method. Second, the blindspot is an absolute oval scotoma, with a one-degree peripheral zone of amblyopia. For this reason, the largest oval white test object that can easily be made to "sink" into the blindspot is chosen for my technique of plotting the blindspot. Individuals vary in this ability, seemingly according to their power of attention and their IQ.

An O-shaped white test object, 50 mm. by 70 mm., has been found convenient in my hands. The narrow white rim facilitates contrast and enhances (by spatial summation) the sensitivity of the retina immediately around the nervehead, and hence, speeds up recognition by the patient. Because of the diminished sensitivity of the retina temporal to the blindspot as pointed out by Wertheim (and also Traquair), the white temporal rim is made twice as wide as the nasal, which measures 1.5 mm. Thus, an illusion of a blindspot enlargement temporally is avoided. The central area, 30 mm. by 50 mm., is not shaded black in order to challenge the pa-

tient's power of fixation as well as to make him realize that the blindspot is completely and actually devoid of perception.

The other side of the test object is all black and nonglaring, and on the two poles and two midsides are small white round test dots. Here, again, the test dot at the temporal side, which measures three mm. in diameter, is made correspondingly larger than the rest. Those on the upper and lower poles measure two mm.; the dot on the nasal side, 1.5 mm.

The large O-shaped test object may be attached to a black nonglaring wand one meter in length, which can be conveniently used in checking the distance between the patient's eye and the fixation point.

TECHNIQUE

His head resting vertically in the headrest and his chin in the chinrest, the patient is seated in front of an ordinary tangent screen so that the eye whose blindspot is to be plotted first is on a level with the fixation target and one meter away. The use of such a device to keep the head in a fixed position, although deplored by some, is not of little importance; for the slightest forward or backward sway will affect the size and location of the blindspot, especially in the horizontal meridian. It is a common observation that even the most co-operative patient cannot keep himself from moving a little during the test. As usually done, the illumination is kept at seven foot-candles, and comes from above and behind the patient. After seeing that the patient is comfortable, the perimetrist instructs the patient to focus on the fixation target, which is a five-mm. white nonshiny disc, bearing a tiny cross at its center.

At this juncture, a short explanation is made to the patient about the presence of a blindspot in the field of each seeing eye. Then the large oval test object (fig. 1) is placed in the center of the blindspot marked out with black thread on the tangent screen, and maintained there, while the eye with whose blindspot we are not concerned at the moment is covered and uncovered. If the patient is



Fig. 1 (Carbajal). Plotting the blindspot in O.S. To show the blindspot in the picture more clearly, the perimetrist had to stand farther from the patient than usual. Note also that the blindspot outlines have been exaggerated.

fixating properly, he will note disappearance and reappearance of the large test object in the blindspot. If the test object is only partly gone, it should be very cautiously moved side to side or up and down until it is completely swallowed up in the blindspot. Also a slight adjustment in the patient's distance from the screen may be necessary to facilitate disappearance of the large test object.

The patient is now instructed to say "yes" as soon as any portion of the white rim (usually seen as a glow) of the test object is made out. It is best to start by allowing the temporal rim to present first. Then the object is moved nasalward on the same horizontal plane and, after disappearing at the center of the blindspot, its nasal rim is slowly allowed to appear. Mental note of the point at which the test object first becomes visible is made, and no markers are used because any interruption at this crucial moment is conducive to a shift in the patient's fixation. Then along the vertical plane passing through the imaginary center of the horizontal meridian that has just been found, the test object is carefully moved to mark out the upper and lower poles of the blindspot. Again, as in the horizontal meridian, the test object should disappear completely between the upper and the lower poles. If this does not happen, the patient is encouraged to maintain fixation on the tiny cross in the central target.



Fig. 2 (Carbajal). Using the other side of the test object.

If there is no generalized enlargement, one may further explore the blindspot by using the other side of the test object (fig. 2). The same technique as in the preceding is followed, with the exception that more stress is placed on the shape and direction of the extensions, if present. Also all four round test dots have to disappear completely in the blindspot area before the test is carried any further. It will be found that the patient having become acquainted with the preceding steps will have little or no difficulty in "sinking" the four round test dots into the blindspot.

The same procedure is done on the other eye (fig. 3) before the periphery of the fields is delineated in the usual fashion. The two blindspots can then be compared and the chances of discrepancy from fatigue are less.

Since the test object used is large, any difficulty in fixation will be quickly spotted as the patient will suddenly see the whole face of the test object or a good part of it. But once this has become swallowed up in the blindspot, the patient can concentrate on fixating the central target. Moreover, the rim of the O-shaped test object, especially temporally, will stimulate more retinal elements than would a single 2/1,000 one. Another advantage of using this large test object is that finer, slower, and fewer excursions (just horizontal and vertical meridians in place of the usual eight) are made possible. Hence, the test is less time-consuming and less tax-

ing to both patient and perimetrist.

It should be borne in mind, however, that this test object is designed primarily for measuring the blindspot. In other types of scotomas, which usually have irregular borders, the ordinary test objects (one mm. to 10 mm.) should be used. Although the above-mentioned test objects are used in mapping out the periphery of the fields, it may be found advantageous to use the large O-shaped test object in cases in which the sensitivity of the retina is markedly diminished as in central retinal vessel occlusion, detached retina, brain tumor, and advanced glaucoma. This test object may also be used in malin-gering to check the size of the blindspot, which should increase at two meters. Where the visual acuity (central vision) is poor as in aphakia, extremely high refractive errors, and in conditions presenting central scotoma, it may also be used as a fixation target.

SUMMARY

1. The average measurements and location of the blindspot on the tangent screen and the factors affecting its size are enumerated briefly.

2. Enlargement of the blindspot may be uniform or localized. Examples of each are cited.



Fig. 3 (Carbajal). Plotting the blindspot in O.D. Take careful note of how the wand and the occluder are held. In order to make the temporal rim of the test object correspond with the temporal side of the screen, the wand has to be held from above when the right blindspot is being examined, and from below for the left.

3. Demonstration of the enlargement of the blindspot is of prime importance in the early diagnosis of papilledema and glaucoma.

4. The reasons for plotting the blindspot before doing the periphery of the fields are reviewed.

5. The current methods of mapping out the blindspot make use of small round white

test objects, rarely larger than 20 to 40 mm. A new O-shaped test object, 50 mm. by 70 mm., for plotting the blindspot, the technique, and advantages of using it, are presented.

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ELECTROPHORETIC STUDIES ON STORED CORNEA*

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Recently we have shown in this laboratory that storage of eyes by freezing, following pretreatment with glycerine, maintains the clarity of the cornea indefinitely, in contrast to storage at 4°C. where it usually becomes cloudy within two weeks.¹ We have shown also that both epithelial cells and fibroblasts remain viable, in about 90 percent of tissues, up to four weeks' storage in liquid paraffin, and up to three weeks in aqueous vapor, in spite of gross cloudiness of the cornea. On the other hand, corneas stored at -79°C. following glycerine pretreatment, and rapid freezing, were not viable after one and one-half hours of storage, although they maintained clarity. Viability was maintained, however, when the rate of freezing was slowed by wrapping the jars containing the eyes in gauze before placing them in the deep-freeze.²

A study of corneal proteins by electrophoresis was undertaken to discover if any changes occurred during storage. It seemed possible that the clouding of corneas stored at 4°C. might be due to denaturation of protein by enzyme action. It is unlikely that these changes would take place at -79°C. since enzyme action would be extremely slow at that temperature.

Electrophoresis has been used in the study of corneal proteins by Smith and Woodin,³ and by François and Rabaey.⁴ Both groups of investigators showed that four protein components could be separated by electrophoresis at pH 7.0. However, since their results were based on studies with bovine eyes using several buffers at different pH, they could not be compared with present studies in which we used rabbits' corneas at

a pH of 8.6 and a barbituric buffer. Moreover, the previous studies were carried out on fresh tissues, whereas the present studies were designed to show changes in protein fractions during storage.

METHODS AND MATERIALS

Eyes were removed from rabbits soon after death, and were immersed in a solution containing penicillin and streptomycin.

Prior to deep freezing, eyes were placed in 15-percent glycerine-saline solution for one hour. All eyes were then placed in individual containers. Some were frozen rapidly in a mixture of alcohol and carbon dioxide snow and stored at -79°C. in a dry-ice chest.⁵ Other containers were wrapped in gauze for slow freezing, and stored in the dry-ice chest at -79°C.⁶ Another series were slow-frozen and stored in a commercial deep freeze at -20° and at -30°C.

Eyes which were not frozen were stored in aqueous vapor, and in liquid paraffin, at 4°C.

PREPARATION OF MATERIAL

The water soluble protein was obtained by extraction from sections of rabbit cornea with distilled water at 4°C. for two days. Insoluble material was eliminated by centrifugation, and the supernatant fluid containing the protein was evaporated in a dry-freezing apparatus. The solid protein was redissolved in distilled water and dialyzed overnight against barbituric buffer at pH 8.6. Electrophoretic studies were carried out on the final solution containing two percent total protein, using the technique of Durrum (Spinco).

The eyes stored by freezing methods were thawed rapidly by placing the jar in a 37°C. water bath, or by pouring normal saline warmed to 37°C. directly over the eyes. The eyes stored in liquid paraffin were washed

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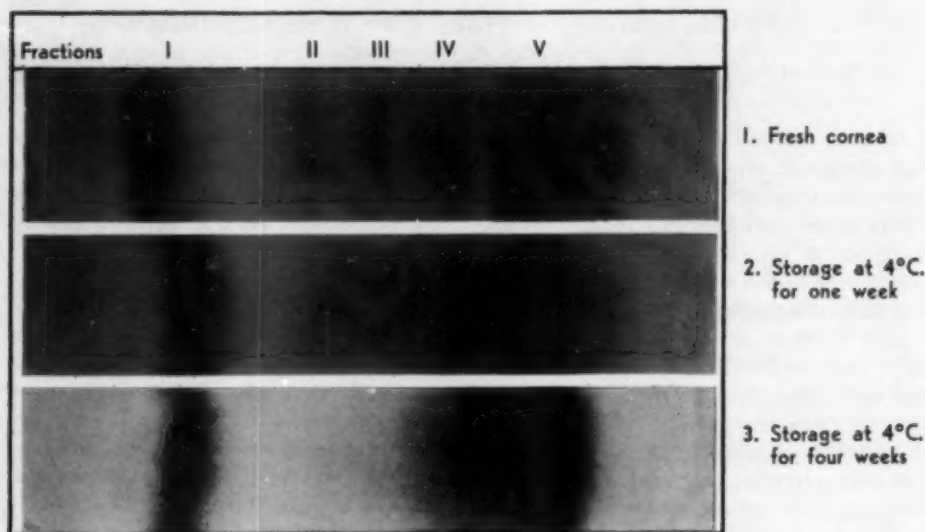


Fig. 1 (Fielding, Basu and Ormsby). Electrophoretic pattern of water-soluble corneal proteins.

several times in normal saline before the corneas were removed.

RESULTS

Separation of the water-soluble proteins of fresh rabbit cornea showed five distinct fractions:

Fraction I travelled with the same mobility as serum albumin.

Fraction II lay between the α_1 and α_2 fractions of serum globulin.

Fraction III lay between the α_2 and β fractions of serum globulin.

Fraction IV lay behind the β fraction of serum globulin.

Fraction V lay in the same region as serum α globulin.

The albumin fraction remained unchanged in all experiments. Alterations which occurred during storage were limited to the globulin group.

Under all the conditions of storage in which the temperature was below 0°C., the electrophoretic pattern was similar to that obtained from fresh cornea. In contrast, the tissue stored at 4°C. both in liquid paraffin and aqueous vapor showed progressive alter-

ation in the proteins after the first week. Further studies of corneas stored at 4°C. at weekly intervals showed a gradual loss of specificity of the protein fractions, so that by the fourth week of storage the four globulins had coalesced into one (fig. 1).

DISCUSSION

The changes in the globulin fractions seen by electrophoresis are of interest, since they first appear at the end of the first week of storage at 4°C., and correspond to the onset of gross cloudiness in the stored corneas. We cannot assess the clinical significance of these findings at this time, since it has been shown in previous studies that both epithelial cells and fibroblasts remain viable in tissue cultures and on the chorioallantoic membrane of the chick for at least two weeks.^{3,7} It is possible that the failure of cloudy donor material to become clear following grafting is more dependent upon protein changes than upon viability of the cells.

The fact that deep frozen corneas do not show protein change by electrophoresis up to three months of storage, and that viability can be demonstrated in these tissues,^{3,7} sug-

gests that further trials with deep frozen donor tissues should be made in corneal grafting.

SUMMARY

1. Rabbits' eyes were stored at 4°C. in aqueous vapor and in liquid paraffin, and in the deep-freeze following glycerine pretreatment. After removal of the eyes from storage, the corneal proteins were extracted and studied by electrophoresis.

2. At temperatures below 0°C., no change in proteins occurred up to three months of storage.

3. Unfrozen corneas stored at 4°C. showed progressive change in the globulin fraction, beginning about the end of first week of storage and corresponded to the onset of gross cloudiness in the stored corneas.

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CATARACT IN RATS FED ON HUMAN MILK*

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The occurrence of lens changes in rats fed exclusively on human milk was reported in 1941 by Gerbasi during research on the anemic state resulting from this kind of diet; the finding was later confirmed by Burgio, but a precise ophthalmologic report was lacking.

Gerbasi, using vitamin dietary supplements, was able to exclude that a deficiency of this kind could be considered responsible for the cataract and instead ascribed the observed changes to the particularly high lactose content of human milk.

The present research was undertaken to

confirm these data, which have remained buried in the literature, in order to throw further light on this interesting kind of experimental cataract.

PROCEDURE

A group of 25 albino rats, about three weeks of age, weighing 30 to 35 gm., was fed over a period of 50 days on human milk supplied at will in glass containers. Slitlamp examination was carried out twice a week, previous pupillary dilatation being effected with a drop of one-percent atropine. In all rats, growth was markedly subnormal, all developed a severe diarrhea which never subsided during the course of the experiment; nine of them died before the end of the study

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Fig. 1 (Vozza). Rat fed for 50 days on cow's milk (above) compared to a rat of the same age (below) fed for the same period on human milk.

of intercurrent lung affections to which they showed particular sensitivity, probably due to a severe pernicious anemia-like state and poor general condition. In fact the animals showed a picture of starvation—ruffled hair, wide areas of alopecia, and loss of vivaciousness, especially in comparison with control rats fed on cow's milk. The mean final body weight was 62 gm.

All rats were killed by bleeding in order to determine blood sugar levels; all eyes and some organs were extracted for histologic examination.

The 10 control rats which had been placed on a cow's milk diet showed subnormal growth and, although anemic, looked lively and healthy and did not exhibit physical defects except subnormal growth which was, however, much better than that of the rats kept on human milk diet (fig. 1). The mean final body weight was 138 gm. which is markedly subnormal for our breeding stock.

Ten rats of the same age and weight were placed on a diet consisting of cow's milk, whose lactose content was brought up to 6.5 percent in order to reproduce the same carbohydrate percentage of human milk; commercial lactose was used for this purpose.

Another group of 15 rats, divided in three cages containing five rats each, received a diet of cow's milk the lactose content of which was brought to 10, 20, and 30 percent respectively. One rat of the 20-percent lactose group died before the end of the experiment of an undetermined cause since it was

partially eaten by the other rats of the cage. All animals showed markedly subnormal development, weak appearance, partial alopecia, ruffled hair, and considerable loss of vivaciousness. All were affected with a severe diarrhea.

Fifteen control rats, also divided in three groups of five each, received a standard diet (Randoin-Causaret), to which lactose was added in the same proportion.* None of these animals showed pathologic processes, except scanty growth and a slight degree of diarrhea in the 30-percent lactose group, which, however, subsided after the first week of treatment.

The mean final body weight was 131 gm. for the 10-percent lactose group; 127 and 120 gm. for the 20-percent and 30-percent lactose groups, respectively.

Finally, in order to test the effects of an increased protein content, two groups of five rats each were treated with casein (20 percent) added to human milk and to cow's milk, plus 20-percent lactose. In both these last groups, growth, although subnormal, was much better than in those fed on diets identical except without casein; all of the other pathologic changes were also noted in these animals but to a lesser degree. The mean final body weight was 80 and 75 gm., respectively.

RESULTS

The results of our experiments are summarized in Table 1.

All rats that survived after 50 days of human-milk feeding developed bilateral cataractous lens changes. The earliest alterations were recorded within 18 to 30 days; in all the animals these changes were observed in the anterior cortex in the form of opacification of the anterior suture line and, with lesser frequency, as small pinpoint opacities, or flakes, or vacuoles, in the subcapsular layer

* RANDOIN-CAUSARET DIET:

Crushed cereals	88 %
Wheat germs	5.0%
Casein	5.0%
Calcium chloride	0.5%
Calcium lactate	5.0%

TABLE 1
RESULTS OF EXPERIMENTS

Treatment	Number of Rats	Dead	Incidence of Cataract	Day of Onset	Final Stage	Mean Final Body Weight (gm.)
Human milk	25	9	$\frac{1}{4}$	24 ± 6	+++	62
Cow's milk	10	2	—	—	—	138
Cow's milk + lactose 6.5%	10	—	$\frac{1}{4}$	33 ± 3	+	78
Cow's milk + lactose 10%	5	—	$\frac{1}{4}$	31 ± 3	+	80
Cow's milk + lactose 20%	5	4	$\frac{1}{4}$	28 ± 6	+	58
Cow's milk + lactose 30%	5	—	$\frac{1}{4}$	16 ± 3	++	59
Standard diet + lactose 10%	5	—	—	—	—	131
Standard diet + lactose 20%	5	—	—	—	—	127
Standard diet + lactose 30%	5	—	$\frac{1}{4}$	30 ± 3	+	120
Human milk + casein 20%	5	—	$\frac{1}{4}$	38	+	80
Cow's milk + lactose 20% + casein 20%	5	—	$\frac{1}{4}$	45	+	75

+ Slight cortical or nuclear opacities.

++ Marked cortical and nuclear opacities.

+++ Severe cortical and nuclear opacities.

at the periphery of the lens (fig. 2). A few days after the onset of the initial alterations in the anterior cortex, the posterior cortex also began to show similar changes.

A more advanced stage was characterized by the involvement of the nucleus which showed a peculiar haze due to the appearance of small pinpoint opacities; further progress of posterior lesions was followed with difficulty owing to the progress of the anterior alterations which showed a marked tendency to coalesce (fig. 3). Generally, the opacifica-

tion process, once established, progressed very rapidly, so that, at the end of the experimental period, about half of the eyes examined showed a complete, mature cataract (fig. 4).

In all rats it was possible to record signs of vascular keratitis; the corneal changes were particularly marked in those rats in which the picture of starvation was more pronounced. Blood-sugar levels were determined by means of the Hagedoorn-Jensen method and resulted in a mean value of 1.39 gm.

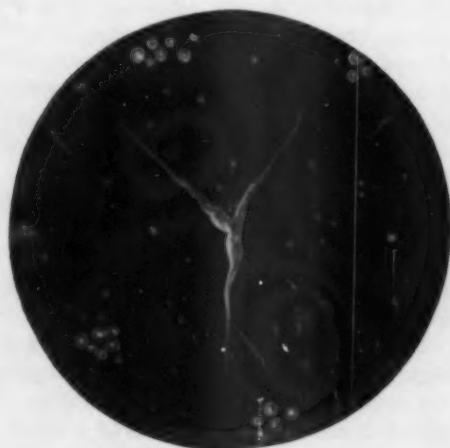


Fig. 2 (Vozza). Human milk cataract (early stage).

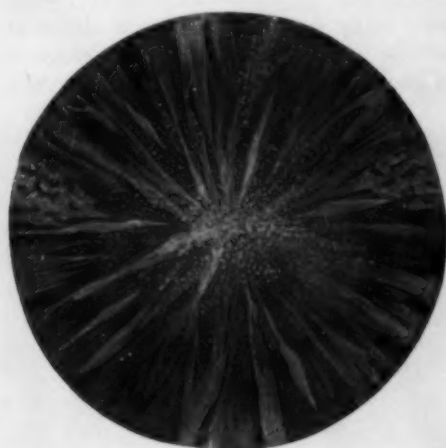


Fig. 3 (Vozza). Human milk cataract (advanced stage).



Fig. 4 (Vozza). Human milk cataract (mature stage).

HISTOLOGIC EXAMINATION

All of the examined lenses showed severe degenerative changes extending to both the cortical and nuclear areas (fig. 5). Everywhere, the cortical epithelium demonstrated a tendency to irregularity in thickness due to marked proliferative activity corresponding to the cortical areas involved in the more severe degenerative changes. A posterior displacement of the nuclear zone and a tendency to the backward extension of the epithelial layer were observed.

Degenerative nuclear processes were sometimes present in the form of pyknosis or decrease of the staining properties. Both in the subcapsular layer and in the cortex, a considerable number of vacuoles of various sizes were found, coupled with liquefied areas filled with an acidophil amorphous or granular material (fig. 6). Fragmented swollen

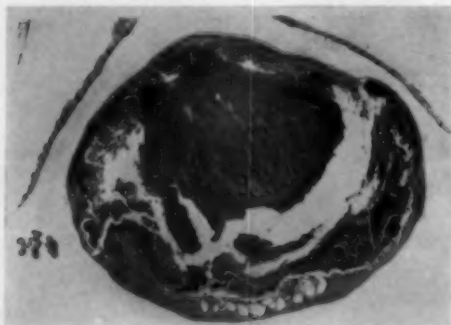


Fig. 5 (Vozza). Histologic picture of mature human milk cataract.

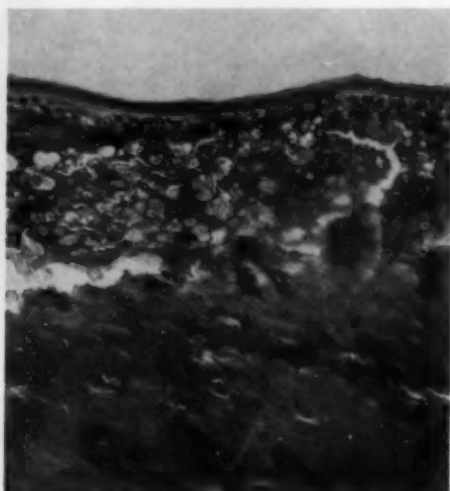


Fig. 6 (Vozza). Section showing severe degenerative cortical changes after 50 days on a diet of human milk.

len fibers sometimes were mixed with newly formed fibers characterized by well-stained round or spindle-shaped nuclei. Even the nuclear area appeared to be affected by degenerative changes but to a lesser extent than the cortex. No other changes were found in the remaining ocular tissues, excepting occasional signs of vascular keratitis, particularly in the peripheral sections.

In the other organs examined, marked changes were observed in the liver, which showed wide areas involved in a severe degenerative process, characterized by fatty degeneration and cloudy swelling (fig. 7). At the periphery of these areas and sometimes in the middle, multiplication of the surviving hepatic cells was observed. The newly formed cells often showed lack of normal lobular arrangement and gave way to a compact tissue. Mitotic stages and binucleated cells were noted.

Throughout the whole intestinal tract a slight, diffuse hyperemic state was present. No cataractous changes of any kind were recorded among the control rats kept on the cow's milk diet; in four out of 10 rats, a slight degree of vascular keratitis was ob-



Fig. 7 (Voza). Section of the liver, showing fatty degeneration and cloudy swelling.

served. The blood sugar level appeared normal, having a mean value of 1.09 gm.

Two out of the 10 rats fed on cow's milk with the lactose content brought to 6.5 percent developed pinpoint opacities of the anterior cortex and perhaps a slight degree of nuclear haze.

An accurate evaluation of the initial changes has always been difficult due to the small size of the eye and, particularly, to the unsteadiness of the animals in front of the slitlamp, in spite of immobilization. In the group of rats kept on the cow's milk diet with the lactose content raised to 10, 20, and 30 percent, the following data were recorded:

In the first group, three out of five rats developed pinpoint cortical and nuclear opacities. In the second group, the same changes from the point of view of intensity were recorded but with a higher incidence (four out of the five surviving rats). Finally, in the third group (30-percent lactose), all five rats demonstrated marked opacities, both cortical and nuclear.

Vascular keratitis was found in about two thirds of the observed eyes after 50 days of treatment. It should be pointed out, how-

ever, that in those groups fed on a cow's milk diet, such severe lens changes were never found as occurred in the same period of time among the animals fed on a human-milk diet.

Among the animals fed on Randoin-Causseret diet plus 10, 20, and 30-percent lactose, opacities were seen only in the 30-percent group. The changes observed were, at any rate, slighter than those obtained in rats fed on cow's milk with the same lactose percentage. In all these animals, the cornea was normal.

Finally, in order to test the effect of a normal protein content, as indicated above, we treated a group of five rats with cow's milk as a basic diet plus casein (20 percent) plus lactose (20 percent). Another group received human milk plus casein (20 percent). In the first group only one out of five rats demonstrated small flakelike opacities in the anterior cortex; in the second, after 50 days, four rats were definitely normal and one showed a doubtful nuclear haze without evident cortical involvement.

DISCUSSION

The observation of cataractous changes in all rats fed on human milk and the lack of any lesion in control animals placed on a cow's milk diet necessitated consideration of the differences in chemical composition in the two kinds of milk in order to identify, if possible, the cataractogenic factor.

The most striking difference on which attention was concentrated, just as Gerbasi's was earlier, was the higher lactose content of human milk (table 2). The lactose content of cow's milk was therefore raised to 6.5 percent in order to determine whether lactose could be responsible for the changes observed. The percentage of rats showing lens opacities was very small; moreover, the severity of the alterations was in no case comparable to that following human milk. Even with higher lactose percentages, although the incidence of the observed changes was higher, severe opacities did not result.

TABLE 2
CHEMICAL COMPOSITION OF HUMAN AND
COW'S MILK

Kind of Milk	Proteins %	Fats %	Lactose %	Ashes %
Human	1.25	3.14	6.5	0.2
Cow's	3.3	3.8	4.95	0.7

The possibility could not however, be ruled out that this might be due to the fact that the quantities of lactose actually ingested by the rats fed a cow's milk basic diet were certainly lower because of the low milk solubility of lactose. An appreciable quantity of the carbohydrate precipitated out on the bottom of the container and only a small fraction of it was eaten by the animals. Many times a day an attempt was made to feed small quantities of the mixture after thorough shaking but each time the animals refused to eat the precipitate. However, it is possible to conclude that a basic diet of cow's milk favors the onset of cataractous changes to a certain extent, if even small quantities of lactose are added. In fact the lens opacities noted in the rats fed the Randoin-Causseret basic diet occurred only after the addition of 30-percent lactose; in the experimental period of 50 days, this represented the minimum limit at which cataractous changes took place, providing that a 20-percent lactose addition failed to produce any lens alteration.

Probably the greater incidence of lens opacities in rats fed on a basic milk diet is related to the low protein content (3.3 percent) in cow's milk and the even lower content (1.25 percent) in human milk. The importance of this factor has been pointed out by Yudkin and Arnold who were able to demonstrate that reduction of proteins causes a marked shortening of the time required to

obtain galactose cataract. This finding has been confirmed by delaying the onset of galactose cataract by a diet high in proteins (Mitchell and Cook, Mitchell, Cook, and Merriam, Henderson and Mitchell), or protein hydrolysates and derivatives (Moore, Henderson, Mitchell and Richie).

Further evidence for this finding was brought out in the present experiment in those rats fed on human milk plus casein (20 percent) and cow's milk plus lactose (20 percent) and casein (20 percent). Without added casein, both diets were certainly cataractogenous but, as a matter of fact, only one rat in each group showed slight alterations. The facts reviewed herein seem to indicate that, since the galactose fraction deriving from the hydrolysis of lactose is a determinant in the genesis of lens opacities (Yudkin and Arnold), it is possible to classify human milk cataract in the group of galactose cataracts. Even on histopathologic grounds it was not possible to differentiate the cataracts produced in the present experiments from the classic descriptions of galactose cataracts by Gifford and Bellows, Dodge, Sasaki, and others.

The onset of lens opacities may, therefore, be considered within the framework of a general metabolic disorder induced by blocking of the normal physiologic utilization of carbohydrates, as Patterson, Fischer, Hörmann, and others have pointed out.

SUMMARY

Cataractous lens changes were obtained in albino rats fed on human milk. Such alterations have been related to the high lactose percentage and low protein content of human milk.

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SEDIMENTATION RATE IN UVEITIS*

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The classification of endogenous uveitis into granulomatous and nongranulomatous types by Woods^{1, 2} has done much to clarify our knowledge and understanding of this disease. It is difficult in some instances to distinguish between these two types of uveitis and this terminology is not accepted by some.³ There is much to support this differentiation of uveitis into granulomatous (primarily an actual infection of the uveal tract) and nongranulomatous uveitis (primarily the result of hypersensitivity to endogenous allergens from infection elsewhere).^{3, 4} Such division is further supported by a study of the sedimentation rate in those individuals with uveitis.

Although the sedimentation rate is not diagnostic of any specific disease, it is of great help in evaluating the presence of infection in the body and the active stages of certain diseases. The settling of the red blood cells is known to be increased particularly in tuberculosis, pelvic inflammatory disease, infected sinuses and teeth, acute rheumatic fever, rheumatoid arthritis, and pregnancy. It is decreased in asthma, hay fever, urticaria, and other allergic states, if infection is not associated. The changes in the blood which affect the sedimentation rate are

mainly in the protein fraction of the plasma. The most important proteins to affect the rate are fibrinogen, gamma globulin, and alpha-two globulin. An increase in these proteins will increase the sedimentation rate. When these proteins are low, the rate is retarded.⁵

Since uveitis is associated with infection elsewhere in the body it was thought that the sedimentation rate might be elevated when this disease state existed, and might be valuable in following the activity of the disease in order that one might know when treatment could safely be stopped. The method of testing the sedimentation rate at William Beaumont Army Hospital is that described by Wintrobe.⁶

The normal for men is 0-10 and for women 0-20 mm. per hour. For the purpose of this study, all men with sedimentation rates between 10-12 and women with rates between 20-22 were discarded from the evaluation in order that the rates, which would be considered elevated, were definite rather than borderline. The individuals used for controls were admissions to the hospital on the Ophthalmology Service and were unselected except for the fact that they did not have uveitis.

Table 1 shows the numbers of individuals with elevated sedimentation rates in three groups of individuals: (a) controls as de-

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TABLE 1
SEDIMENTATION RATE IN UVEITIS

	Normal Sedimentation Rates		Elevated Rates		Total Cases
Nongranulomatous iritis and iridocyclitis	6	(25.0%)	18	(75.0%)	24
Granulomatous uveitis	13	(86.7%)	2	(13.3%)	15
Controls	93	(84.5%)	17	(15.5%)	110

scribed above, (b) individuals with anterior nongranulomatous uveitis; and (c) individuals with granulomatous posterior uveitis with or without associated anterior uveitis.

The total number of persons with only iritis or iridocyclitis was 31, but only those who were considered to have endogenous, nongranulomatous iritis are listed. Of the seven individuals not included three had a traumatic iritis, two had a keratoconjunctivitis, one had a lens-induced uveitis, and the other had an anterior granulomatous uveitis, possibly associated with sarcoid. All these individuals had normal sedimentation rates.

As can be seen from the chart, 75 percent of individuals with nongranulomatous uveitis had an elevated sedimentation rate, whereas 13.3 percent of individuals with granulomatous uveitis and 15.5 percent of the control individuals had elevated rates. Analysis of the data by the Chi square method reveals the difference in the nongranulomatous and the granulomatous uveitis groups and the difference between the nongranulomatous uveitis and the controls is shown to be highly significant, the level of significance being greater than 0.001 in each case.

In about one half of those persons with nongranulomatous uveitis, the sedimentation rate remained elevated even after their ocular disease cleared. The use of antibiotics and steroids in some of the patients did not alter the rate.

COMMENT

Zwiazuer⁶ emphasizes the need for systemic studies in iridocyclitis and iritis. He has observed increased vascular permeability, acceleration of the sedimentation rate, and increased serum globulin in individuals with iritis. The striking difference in the sedimentation

rate in individuals with granulomatous and nongranulomatous uveitis supports the contention that nongranulomatous uveitis is associated with systemic infection and hypersensitive reaction in the eye.

This hypersensitivity is probably not an allergy in the usual sense of the word since the sedimentation rate is normally retarded in individuals with hay fever, asthma, and other exogenous allergic states. This hypersensitivity is probably of a nature similar to that which one finds in the rheumatic and collagen diseases in which there is an increase in the antistreptolysin titer, as there is in iritis.^{7, 8}

Most of the elevated sedimentation rates were in the intermediate range rather than in the markedly increased range. This indicates a low-grade chronic infection. The normal percentage of elevated sedimentation rates in individuals with active granulomatous uveitis suggests that no active systemic infection is present and that a local infectivity of the eye causes the ocular lesions. This local infectivity of the eye, however, is not strong enough to alter the serum proteins of the blood and consequently the sedimentation rate tends toward normality.

CONCLUSIONS

The percentage of elevated sedimentation rates in individuals with anterior nongranulomatous uveitis is significantly higher than in individuals with granulomatous uveitis. This would strongly support the differentiation of uveitis into granulomatous and nongranulomatous uveitis and further the contention that these two diseases have different etiologies.

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ARE AQUEOUS HUMOR DYNAMICS INFLUENCED BY AGING?*

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The availability of the Mueller electronic tonometer and the investigations by Moses and Bruno¹ and by Grant^{2,3} have called the attention of numerous investigators to the dynamics of aqueous humor elimination. Attempts have been made to find whether age has a bearing on aqueous humor production (F) and facility of outflow (C) in normotensive eyes.

First to study this relation seems to have been Grant² (1950). He stated that age is not a factor in determining the normal values of C and F. DeRoeth and Knighton⁴ (1952) came to the same conclusion. Spencer, Helmick, and Scheie⁵ (1955) performed tonography on a series of subjects, from 11 to 90 years of age, with normal eyes. Outflow studies were performed on 20 normal eyes of

subjects in each decade from 11 to 70 years and on 20 normal eyes of subjects from 70 to 90 years of age. Spencer and associates found that the slight decrease in C and F in patients past the age of 50 years was not significant.

Goldmann⁶ (1951) arrived at opposite conclusions after investigating with his fluorometric test the resistance to outflow (R) of 24 normal subjects divided according to age into two groups: The first included subjects up to 50 and the other those over 50 years of age. Goldmann found an increased resistance to outflow as a function of age. R being equal to $1/C$, it would seem that C values decrease after the age of 50 years.

Recently (1956) Weekers, Watillon, and de Rudder⁷ studied tonographically the resistance to outflow of 90 normal subjects divided into three age groups: under 35, 35 to 55, and over 55 years of age. Their results were well in agreement with those of Goldmann.

Because of such conflicting opinions it was considered that a greater number of subjects with normal eyes should be studied. The purpose of our work, therefore, was to determine whether there is any relation between

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age, intraocular pressure (IOP), facility of outflow (C), and production of aqueous humor (F).

METHOD

We studied 345 subjects with no history or signs of glaucoma, representing a total of 666 normal eyes without marked ametropia (that is, less than two diopters myopia or hypermetropia). The ages of our subjects ranged from 14 to 92 years. One hundred and five subjects (203 eyes) were Negroes and 240 (463 eyes) were Caucasians. According to the findings of Boles-Carenini, Buten, Spurgeon, and Ascher,⁹ there is no significant difference between IOP, C, and F values of Negro and white persons.

Of our subjects, 169 (328 eyes) were female and 176 (338 eyes) were male. Goldmann and deRoeth and Knighton found that sex is not a factor affecting the normal values of C and F. Facility of outflow and rate of aqueous humor elimination were determined by tonography. The techniques and calculations were the same as used in our previous studies and extensively described in a paper by one of us (B. B. C.).⁸ All determinations were performed between the hours of 8:30 A.M. and 10:30 A.M.

All data were analyzed statistically, using the F-test as the necessary preliminary, the t-test to compare averages when the standard deviations did not differ significantly, and the Cochran-Cox test to compare averages when the standard deviations did differ to a significant extent.¹⁰

RESULTS

First we studied the values of IOP, C, and F, grouping the subjects according to age, from 10 to 29, 30 to 49, 50 to 69, and 70 to 92 years of age. Tables 1, 2, and 3 show the values of IOP, C, and F, and the relative frequencies for the four groups. At the end of each column appears the total number of eyes studied (N), average values (M), and standard deviations (s). In Figures 1 to 3 these data are presented graphically, the abscissas corresponding to the tonographic readings and the ordinates to the frequencies.

TABLE 1
AGE AND INTRAOCULAR PRESSURE

Intraocular Pressure (mm. Hg.)	10-29	30-49	50-69	70-92
11.0-13.0	—	—	—	1
13.1-15.0	1	2	3	7
15.1-17.0	4	20	19	14
17.1-19.0	6	25	25	13
19.1-21.0	12	40	55	24
21.1-23.0	25	46	40	27
23.1-25.0	18	47	38	28
25.1-27.0	14	35	35	18
27.1-29.0	1	5	10	8
N	81	220	225	140
M	22.22	21.76	21.68	21.47
s	2.92	3.26	3.37	3.80

TABLE 2
AGE AND FACILITY OF OUTFLOW

Facility of Outflow (cmm./min./mm. Hg)	10-29	30-49	50-69	70-92
0.090-0.150	—	5	12	13
0.151-0.210	27	43	38	46
0.211-0.270	32	58	59	31
0.271-0.330	10	75	57	23
0.331-0.390	8	14	31	10
0.391-0.450	3	13	12	8
0.451-0.510	—	8	10	6
0.511-0.570	1	3	3	2
0.571-0.630	—	—	1	1
0.631-0.690	—	1	1	—
0.691-0.750	—	—	1	—
N	81	220	225	140
M	0.2496	0.2790	0.2851	0.2550
s	0.0731	0.0879	0.101	0.1025

TABLE 3
AGE AND RATE OF FLOW

Production of Aqueous Humor (cmm./min.)	10-29	30-49	50-69	70-92
1.30- 2.60	1	2	7	9
2.61- 3.90	24	52	44	41
3.91- 5.20	33	68	75	37
5.21- 6.50	14	53	52	28
6.51- 7.80	6	24	19	14
7.81- 9.10	1	13	14	8
9.11-10.39	1	5	8	2
10.40-11.70	1	3	6	1
N	81	220	225	140
M	4.743	5.253	5.336	4.866
s	1.55	1.79	1.98	1.85

The average values are indicated by vertical dotted lines.

We thought, moreover, that it might be worth while to analyze our data by grouping the subjects as Goldmann did, that is, into two groups, one up to and including 50 years of age and the other over this age. In Tables 4, 5, and 6 are reported the values of IOP, C, and F and the frequencies of these two groups. The abbreviations at the ends of the columns are the same as in the foregoing tables. In Figures 4, 5, and 6 these data are presented graphically.

The results of the statistical comparisons of IOP, C, and F values for the various age groups are summarized in Tables 7, 8, and 9. The abbreviation NS means "not significantly different at the five-percent level," S

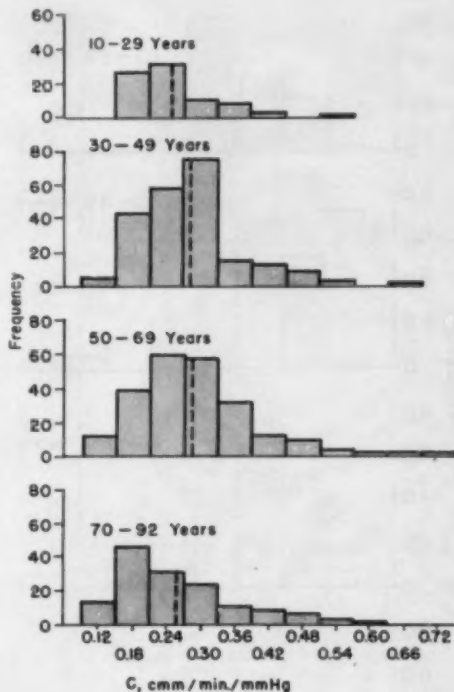


Fig. 2 (Boles-Carenini and Cambiaggi). Variation of outflow facility with age.

means "significantly different at the five-percent level," S* means "significantly different at the one-percent level."

INTRAOCULAR PRESSURE

Application of the t-test or the Cochran-Cox test shows that there is no statistically significant difference between the average intraocular pressures of any of the age groups studied. Table 1 shows, in fact, that the average intraocular pressure falls off slightly with increasing age, but these differences can be attributed solely to chance effects.

Similarly, the average intraocular pressure is not significantly different for the 50 to 92 year group from that for the 10 to 49 year group.

FACILITY OF OUTFLOW

Application of the t-test or the Cochran-Cox test shows that with two exceptions there are significant differences between the

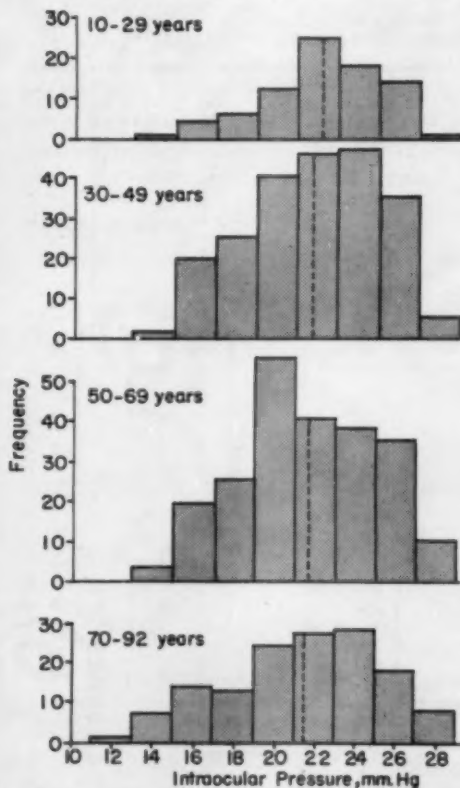


Fig. 1 (Boles-Carenini and Cambiaggi). Variation of intraocular pressure with age.

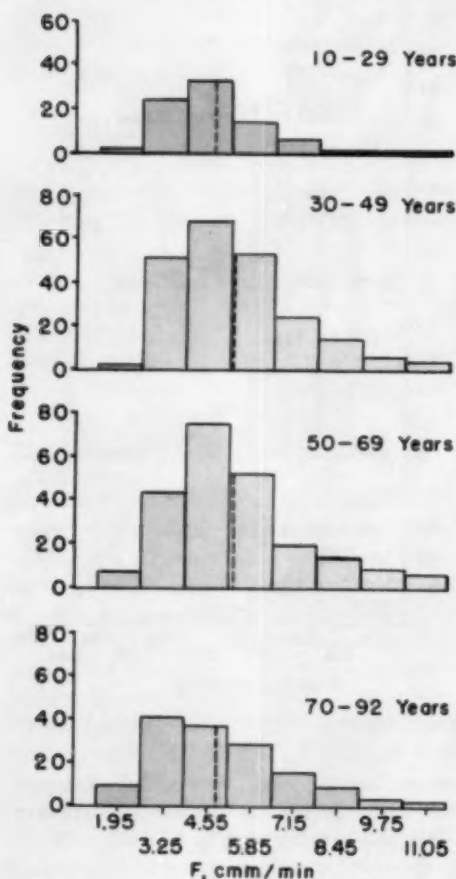


Fig. 3 (Boles-Carenini and Cambiaggi). Variation of rate of flow with age.

average outflow facilities of the various age groups studied. The average outflow facility increases from 0.2496 for the 10 to 29 group, to 0.2790 for the 30 to 49 group, to 0.2851 for the 50 to 69 group and then falls off sharply to 0.2550 for the 70 to 92 group. Because of this sharp decrease the average values do not differ significantly when the 10 to 29 and 70 to 92 groups are compared, or when the 30 to 49 and 50 to 69 groups are compared. All of the other comparisons do show significant differences.

When the 10 to 49 and 50 to 92 groups were compared, the average outflow facilities

TABLE 4
AGE AND INTRAOCULAR PRESSURE

Intraocular Pressure (mm. Hg)	Age (yr.)	
	10-49	50-92
11.0-13.0	—	1
13.1-15.0	3	10
15.1-17.0	24	33
17.1-19.0	31	38
19.1-21.0	52	79
21.1-23.0	71	67
23.1-25.0	65	66
25.1-27.0	49	53
27.1-29.0	6	18
N	301	365
M	21.89	21.60
s	3.17	3.54

TABLE 5
AGE AND FACILITY OF OUTFLOW

Facility of Outflow (cmm./min./mm. Hg)	Age (yr.)	
	10-49	50-92
0.090-0.150	5	25
0.151-0.210	70	84
0.211-0.270	90	90
0.271-0.330	85	80
0.331-0.390	22	41
0.391-0.450	16	20
0.451-0.510	8	16
0.511-0.570	4	5
0.571-0.630	—	2
0.631-0.690	1	1
0.691-0.750	—	1
N	301	365
M	0.271	0.274
s	0.0851	0.102

TABLE 6
AGE AND RATE OF FLOW

Production of Aqueous Humor (cmm./min.)	Age (yr.)	
	10-49	50-92
1.30-2.60	3	16
2.61-3.90	76	85
3.91-5.20	101	112
5.21-6.50	67	80
6.51-7.80	30	33
7.81-9.10	14	22
9.11-10.39	6	10
10.40-11.70	4	7
N	301	365
M	5.116	5.156
s	1.74	1.94

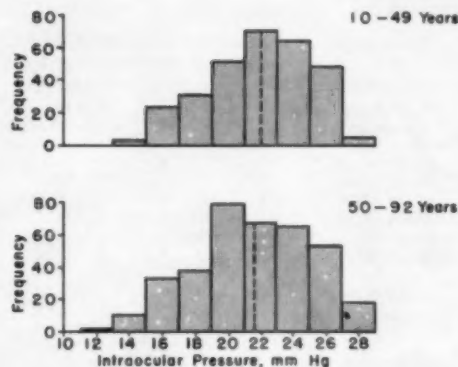


Fig. 4 (Boles-Carenini and Cambiaggi). Variation of intraocular pressure with age (two groups).

are found to be not significantly different. This is due to the fact that the 0.2496 value is balanced by the 0.2550 value, and the 0.2790 value by the 0.2851 value.

When it is considered that the average outflow facility for the 70 to 92 group is still greater than for the 10 to 29 group, it appears that aging, in itself, does not cause an increase in the average resistance to outflow above that of the youngest group studied.

RATES OF FLOW

Similar considerations apply when the

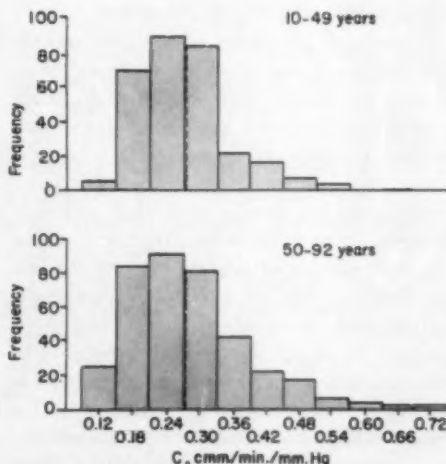


Fig. 5 (Boles-Carenini and Cambiaggi). Variation of outflow facility with age (two groups).

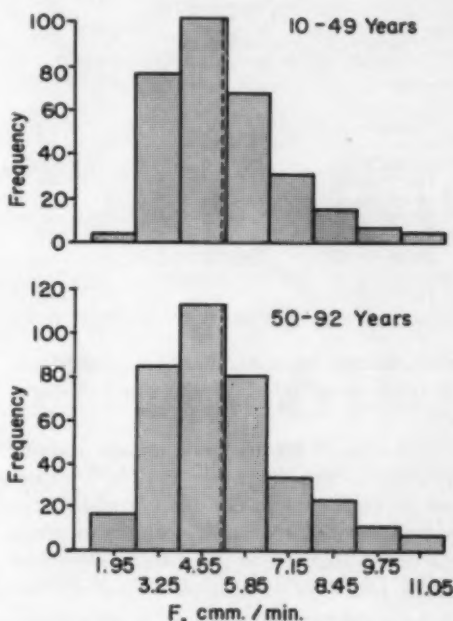


Fig. 6 (Boles-Carenini and Cambiaggi). Variation of rate of flow with age (two groups).

average flow rates are studied. The average flow rates increase with increasing age and then fall off again for the oldest group (table 3). The average rate for the 70 to 92 group, however, is still greater than for the 10 to 29 group. Significant differences are found between the means except when the 10 to 29 group is compared with the 70 to 92 group, and the 30 to 49 group with the 50 to 69 group.

Comparing the 10 to 49 group with the 50 to 92 group, the means of the flow rates are found to differ by an amount so small as to be attributable to chance.

It appears, therefore, that aging, in itself, does not cause a decrease in flow rate below that of the youngest group studied.

DISCUSSION

According to Grant, to deRoeth and Knighton, and to Spencer, Helmick, and Scheie, there is no relation between age and C or F. Our results are well in agreement

TABLE 7

STATISTICAL COMPARISON OF INTRAOCULAR PRESSURES OF DIFFERENT AGE GROUPS

Age Groups Compared	F Test	t Test	Cochrane-Cox Test
10-29/30-49	NS	NS	—
10-29/50-69	NS	NS	—
10-29/70-92	S*	—	NS
30-49/50-69	NS	NS	—
30-49/70-92	S	—	NS
50-69/70-92	NS	NS	—
10-49/50-92	S	—	NS

with theirs, but contradict the conclusions of Goldmann, and of Weekers and co-workers.

The lack of relation between age and our average tonographic values may be interpreted as evidence that the more frequent appearance of glaucoma in elderly patients is not due to anatomic or physiologic alterations in the pathways of aqueous humor elimination caused by aging.

This conclusion is not in agreement with the findings of Teng, Paton, and Katzin¹¹ who described degenerative changes in the vicinity of the chamber angles of normal eyes, not previously reported in the literature. This degeneration probably begins in the external trabecular meshwork and spreads along these fibers to Schlemm's canal, to its outlets, and sometimes to the intrascleral plexus. The cause and nature of this degeneration is unknown, but degeneration of collagenous and elastic fibers has

TABLE 8

STATISTICAL COMPARISON OF OUTFLOW FACILITIES OF DIFFERENT AGE GROUPS

Age Groups Compared	F Test	t Test	Cochrane-Cox Test
10-29/30-49	S	—	S*
10-29/50-69	S*	—	S*
10-29/70-92	S*	—	NS
30-49/50-69	S	—	NS
30-49/70-92	S	—	S
50-69/70-92	NS	S*	—
10-49/50-92	S*	—	NS

TABLE 9

STATISTICAL COMPARISON OF RATES OF FLOW OF DIFFERENT AGE GROUPS

Age Groups Compared	F Test	t Test	Cochrane-Cox Test
10-29/30-49	NS	S	—
10-29/50-69	S*	—	S*
10-29/70-92	S	—	NS
30-49/50-69	NS	NS	—
30-49/70-92	NS	S	—
50-69/70-92	NS	S	—
10-49/50-92	S	—	NS

been demonstrated. Due to adhesion and proliferation of endothelium, caused by degenerative processes, the drainage channels were obliterated. The incidence of this primary degeneration was found to be higher after the age of 40 years.

Ashton and associates,¹² however, stated that it is difficult to assess the validity of the conclusions of Teng and associates, especially because similar anatomic changes may be attributed to artifact or to post-mortem changes.

Dvorak-Theobald and Kirk¹³ concluded (1956) from microanatomic study that hypertrophy and sclerosis of the collagenous scleral fibers associated with aging can produce narrowing or occlusion of the intrascleral veins, resulting in glaucoma.

If anatomic alterations of the aqueous outflow pathways due to aging really occur in all eyes but, as we have demonstrated, there is no relation between age and average tonographic readings in normal eyes when the oldest and youngest age groups are compared, then we can assume that anatomic changes are not likely to alter the aqueous humor dynamics of normal eyes or else are compensated for by particular physiologic factors such as opening of new outlets or changes in episcleral venous pressure. In glaucomatous eyes, however, the trabecular meshwork degeneration or changes in the scleral collagenous tissues may be more severe, enough to influence the aqueous outflow

directly, or the compensating factors may have become inadequate.

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ACKNOWLEDGMENT

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LEPROUS IRITIS WITH HYPOPYON*

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Leprosy frequently involves the eye. Various investigators have reported that ocular complications may occur in 90 cases out of 100 (Prendergast,¹ Harley,² Kennedy³).

The cornea is most frequently affected; the anterior segment is next. The iridocyclitis is usually plastic in type; nodular iridocyclitis is less frequent; while the serous type rarely appears (Kalt⁴).

Leprous iritis with hypopyon is seldom met and only a limited number of cases have been recorded. No previous laboratory studies of the aqueous humor can be found in the literature except in the case of Voisin and

Lombard⁵ which was presented before the Ophthalmological Society of Paris in November, 1954. Another case was presented by me⁶ before the Congress of the French Ophthalmological Society in May, 1956.

Voisin and Lombard reported an old leprous iritis with hypopyon associated with a severe keratitis and a chronic purulent dacryocystitis. These authors twice performed a puncture of the anterior chamber and the aqueous humor was examined according to the method of Amsler and Verrey.

The result of the first examination revealed an abundance of red blood cells, lymphocytes, polymorphonuclear and iris cells. Ziehl-Nielsen's staining method showed

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Hansen's bacilli, some of which were intracellular. In the second puncture no leprosy mycobacterium was identified. I pointed out that anterior chamber puncture revealed the following interesting features: the albumino-cellular concordance, the preponderance of lymphocytes (more than 80 percent), and the complete absence of Hansen's bacilli. Finally I reported the spectacular therapeutic effect of hydrocortisone acetate, with the hypopyon disappearing in 24 hours.

Recently, I have had occasion to study two more cases of leprosy iritis with hypopyon. The patients were from the leprosarium of St. Barbara in the Athens area where more than 500 patients are being treated. Two patients were under observation and treatment, and the aqueous humor obtained after a puncture of the anterior chamber was examined in the Eye Clinic of Athens University.

It was possible to study the course of the ocular disease from the day it first appeared. In both cases hypopyon iritis was the first ocular manifestation of the disease. I studied aqueous humor obtained through needle puncture of the anterior chamber during the course of the disease. The study consisted of (1) quantitative examination of the leukoma (Pandy's reaction); (2) cell enumeration; (3) differentiation of the cell features contained in aqueous humor stained according to the May-Grünwald-Giemsa method (modified by Verrey); (4) Ziehl-Nielsen's stain in a search for acid-resistant bacilli. Specimens taken from aqueous humor were cultured on Loewenstein's medium for the same purpose.

In the second case, the aqueous samples were injected into the anterior chamber of rabbit eyes. The clinical and histochemical studies permit reporting some significant observations, one of which was that topical application of corticosteroids (hydrocortisone acetate) as eyedrops and by subconjunctival injections seemed the only way to treat both cases, permitting an estimate of the great therapeutic value of the drug.

REPORT OF CASES

CASE 1

A man, aged 38 years, had been hospitalized for five years with a diagnosis of tuberculous leprosy. Three years ago he was treated, not systematically, with sulfone drugs because of severe digastric disturbances. For the last eight months this treatment had been abandoned. Previously he had no ocular manifestations of leprosy. On May 20, 1956, he came for examination complaining of acute pain in the right globe, redness, photophobia, and blurred vision. These symptoms were accompanied by severe headache.

Clinical examination of this eye revealed severe pericorneal injection, a slight pupil retraction, and a hypopyon about two-mm. in height.

Biomicroscopic examination revealed an intensively positive Tyndall phenomenon (three plus). Examination failed to reveal other pathologic findings in either the cornea or iris. Especially no deposits were noted on Descemet's membrane.

The hypopyon should be described because it differs from hypopyons of other causes. In this case the hypopyon was much more fluid, the color nearly white, and the surface was flat instead of fingernail-shaped, as is usual.

The visual acuity of the eye was 1/10. The intraocular pressure was slightly decreased. The patient complained that the globe was painful when touched. The other eye was entirely normal.

The anterior chamber was punctured just above the surface of the hypopyon, according to the method of Amsler and Verrey, and aqueous humor was obtained, part of which was examined. The rest was used for specimens cultured on Loewenstein medium. Direct pupil mydriasis was with atropine eyedrops, adrenalin was injected subconjunctivally, and 20 mg. of hydrocortisone acetate were injected near the limbus. Hydrocortisone eyedrops (one percent) each hour and one-percent atropine solution twice daily were ordered.

From the histochemical examination of the aqueous humor it was learned that (1) the quantity of albumin counted by the Pandy reaction was abundant (three plus); (2) cell count (Malassez's cellule) revealed 224 cells per c.mm.

The morphology of the aqueous humor cells examined after capillary centrifugation and after staining showed: lymphocytes and lymphocytoides, 86 percent; cells of the reticulo-endothelium system, eight percent; iris cells, two percent; polymorphonuclear leukocytes, two percent, degenerated cells, two percent. Hansen's bacilli stained by Ziehl-Nielsen's method were negative.

Course. The next day, after the first examination, spectacular improvement, both subjective and objective, was noted. The patient was no longer complaining of headache and the pain in the eye was less acute. Photophobia had completely disappeared. The vision had improved to 3/10. The Tyndall phenomenon was intensively positive. The pupil was completely dilated and the intraocular

pressure was 28 mm. Hg. The fundus picture was not very clear but no pathologic features were found. Continuation of the hydrocortisone drops every hour and atropine once daily were ordered. There was progressive improvement over the following days. Vision had improved. The intraocular pressure fluctuated between 30 and 20 mm. Hg.

Three days after the appearance of the disease, subconjunctival hydrocortisone acetate (20 mg.) was administered near the limbus. During the following week hydrocortisone (one percent) was instilled every hour and atropine daily.

On May 27, 1956, seven days after the appearance of the disease, there was no other subjective sign except slightly blurred vision. The examination showed at some point an extremely light circumcorneal injection. The Tyndall phenomenon was still intensively positive. The pupil was completely dilated. The intraocular pressure was 22 mm. Hg and the vision had improved to 8/10. The puncture of the anterior chamber revealed a three-plus Pandy, 160 cells per mm., 82-percent lymphocytes and lymphocytoides, five-percent reticulo-endothelial cells, eight-percent iris cells, three-percent polymorphonuclear leukocytes, two-percent degenerated cells, Hansen's bacilli (Ziehl's stain) were negative. Subconjunctival injection of hydrocortisone acetate (20 mg.) was repeated. During the second week, hydrocortisone (one percent) every two hours and atropine eyedrops every 24 hours were continued. Subconjunctival hydrocortisone (20 mg.) was administered twice a week. The condition of the eye continued to improve.

On June 2, 1956, the eye was nearly quiet, the vision had improved to about 10/10, the intraocular pressure was 22 mm. Hg, and Tyndall one plus. At this time a puncture of the anterior chamber showed a two-plus Pandy and 80 cells per c.mm. The study of cells revealed about the same percentage of the various types as in the previous examinations. Hansen's bacilli were negative.

During the first month of the disease the patient continued on treatment with local hydrocortisone drops and subconjunctival injection of hydrocortisone twice a week. Atropine was instilled as before. The condition of the eye continued to improve. The vision improved to 10/10 and the intraocular pressure had fallen to the normal limits (20 mm. Hg). On June 20, 1956, Tyndall was one plus for the first time; four or five small white deposits on Descemet's membrane were noted. The vision was normal, 10/10; the intraocular pressure was also within normal limits. The puncture of the anterior chamber revealed a one-plus Pandy, 32 cells per c.mm. and predominative lymphocytes, as in previous examinations. No bacilli of Hansen were found.

During the second month therapy with hydrocortisone drops was continued three times daily and subconjunctival hydrocortisone (20 mg.) once a week. The condition of the eye continued to improve.

On July 20, 1956, no other clinical inflammatory

signs were noted except the deposits on Descemet's membrane equal to the number in the previous examination and now a little colored. The vision was normal; the intraocular pressure was normal, too. The examination of the aqueous humor at this time revealed a one-plus Pandy reaction, 16 cells per c.mm., two thirds of which were lymphocytes and lymphocytoides and the remaining one third reticulo-endothelial and iris cells.

On August 20, 1956, although no clinical signs were noted, the reaction of Pandy remained slightly positive. There were nine cells per c.mm. During the third month, therapy with hydrocortisone drops twice daily was continued. Hansen's bacilli were negative both on microscopic examination and in the specimens cultured on Loewenstein's medium.

CASE 2

A woman, aged 28 years, had been hospitalized two years with a diagnosis of tuberculous leprosy. She was systematically treated with sulfone drugs with satisfactory results as far as her general health was concerned. She had never had any ocular manifestations until three days prior to her examination.

She was first seen on June 5, 1956, complaining of an acute pain in her right globe, an intensive photophobia for the previous three days, blurred vision, and a red eye. All these signs were accompanied by intensive headache. During these days she had been given various analgesics, penicillin drops in her painful eye three times daily, and cortisone eyedrops, without relief. The condition of the eye continued to become worse.

Clinical examination revealed a slight swelling of the eyelids and a very intensive hyperemia of the globe. The cornea was absolutely clear; no deposits of Descemet's membrane were observed. The hypopyon in the anterior chamber was fluid, its color nearly white, and its flat, free surface reached about one mm. under the lower pupillary lid. The pupil was in miosis and reacted slightly to light. The Tyndall phenomenon above the hypopyon was intensively positive (three plus); the vision was 1/40. The intraocular pressure was a little increased and the globe was painful when touched. The other eye was normal.

A puncture of the anterior chamber was performed above the free surface of the hypopyon and 0.3 cc. of not very clear aqueous humor was obtained. The puncture was performed with difficulty because of her pain. Since cocaine (four percent) did not give sufficient anesthesia, four-percent novocaine with adrenalin was injected subconjunctivally and a retrobulbar injection of the same solution was given. After the puncture, hydrocortisone (30 mg.) was injected subconjunctivally near the limbus and atropine eyedrops were instilled. Hydrocortisone eyedrops every half hour and atropine twice daily were ordered.

Study of aqueous humor revealed a four-plus Pandy and 486 cells per c.mm., with 80-percent

lymphocytes and lymphocytoides, 10-percent reticulo-endothelium and iris cells, and 10-percent degenerated cells. Hansen's bacilli (stained by Ziehl-Nielsen method) were negative. Specimens were cultured on Loewenstein medium.

Rabbit inoculation. A small amount of aqueous humor was inoculated into the anterior chamber of the eye of a rabbit. On the first day slight inflammatory signs of the anterior segment were observed (pericorneal injection, Tyndall positive, and a slight miosis of the pupil).

Nine days after the inoculation, no inflammatory signs were observed clinically or in the laboratory. In fact, after puncture of the anterior chamber of the rabbit, no pathologic signs were found in the aqueous humor. The eye of the rabbit was followed for two and a half months after the inoculation was performed without any pathologic features being observed.

Observation of the patient's eye showed the following:

The next day a satisfactory improvement was noted, the hypopyon had decreased, and the vision had improved to 1/10.

Near the place where the hydrocortisone injection was performed, an excavation of the cornea was observed in the cornea and at a distance of one mm. from the limbus. Its shape was an irregular circle about 3.0 to 4.0 mm. in diameter. The surface of the whole excavation was regular, its depth reached half of the thickness of the cornea. A solution of methylene blue (one percent) was instilled and produced only slight coloration.

Skilamp examination showed the excavation to be covered with epithelium. The photophobia of this eye was less than on the first day, the patient was no longer complaining of pain. Hydrocortisone eyedrops every two hours and atropine twice daily were ordered.

Course. The following days the eye improved a little. The hypopyon always had the same form and the same color, its height being about 1.5 mm. The vision was 2/10. The excavation of the cornea remained the same. The same treatment was continued. On June 7, 1956, the height of the hypopyon was 1.5 mm.; it was fluid, rather white, its surface was flat, Tyndall was three-plus, and the excavation of the cornea was smaller. The vision remained at 2/10; the intraocular pressure was lower than that of the normal eye. Hydrocortisone acetate (25 mg.) was injected at a point opposite the previous injection. The following day hypopyon had completely disappeared, Tyndall remained intensively positive, the visual acuity had improved to 3/10. The photophobia and the swelling of lids had disappeared. The hyperemia of globe was less. Only at one point where hydrocortisone was injected was there swelling and intensive hyperemia. Near the place of the second injection, a new excavation of the cornea was observed, having the same dimension and the same characteristics of the first one. The excavation remained as at the previous examination.

On June 10, 1956, the lids were normal. The

photophobia was no longer present. Circumcorneal injection was rather slight. The first excavation which had appeared in the cornea had nearly disappeared; the second one remained intensively positive. The intraocular pressure was normal. The vision was 3/10.

Puncture of the anterior chamber revealed a three-plus Pandey, 280 cells per c.mm.; the percentage of each kind remained the same as on previous examination. The Ziehl stain revealed no Hansen's bacilli. A subconjunctival injection of hydrocortisone (20 mg.) was given far from the limbus.

The second week the eye continued to improve. The second excavation which had appeared in the cornea had completely disappeared five days after it was first seen, without leaving any opacity. The first excavation gave the same result. Treatment with hydrocortisone acetate (one percent) every two hours and subconjunctival injections (20 mg.) twice every week far from the limbus were continued. On June 17, 1956, visual acuity was 6/10, intraocular pressure 18 mm. Hg, a few thin white deposits were on Descemet's membrane, one-plus Tyndall. Examination of the cornea revealed nothing. Anterior chamber puncture revealed 156 cells per c.mm., lymphocytes and lymphocytoides were in great abundance, Pandey's reaction was intensively positive, Ziehl's stain revealed no Hansen's bacilli.

During the next 15 days, the same therapy was continued. The cornea appeared to be normal. The eye showed progressive improvement. On July 3, 1956, a month after the start of the treatment, the eyelids were clinically normal. In the place where the subconjunctival injections had been performed good-sized yellowish nodules were observed on the conjunctiva which otherwise showed a smooth surface. The few deposits on Descemet's membrane were small, thin, and slightly colored. The places where the excavations had been were no longer visible. Tyndall phenomenon was slightly positive, the pupil round and completely dilated, the intraocular pressure normal.

Careful biomicroscopic examination of the iris revealed no pathologic features. No pathologic features were observed in the lens, vitreous, or fundus. The vision had improved to 10/10.

Laboratory examination of the aqueous humor revealed a two-plus Pandey, 56 cells per c.mm., 72-percent lymphocytes and lymphocytoides, 28-percent reticulo-endothelial and iris cells; also noted were a small number of polymorphonuclear leukocytes and a few degenerated cells whose origin could not be found. No Hansen's bacilli were observed. The second month the same treatment was continued and the condition of the eye improved.

On August 3, 1956, clinically the conjunctival nodules were observed at every point where an injection of hydrocortisone had been given. The patient no longer complained of anything. No other clinical signs of inflammation were observed except for a few slightly colored deposits on Descemet's membrane. Laboratory examination of the aqueous humor revealed a one-plus Pandey, 18 cells

per c.mm., and the percentage of various types of cells as at previous examinations. There was always an intensive lymphocytosis. Bacilli of Hansen were not found either on microscopic examination or on culture.

COMMENT

Two cases of leprous iritis with hypopyon have been completely studied from the first day of the appearance of the disease to complete recovery after more than two months. In both cases the subjective and objective symptoms of the disease and the laboratory studies were the same. The clinical studies of the patients, the laboratory examination of the aqueous humor obtained by puncture of the anterior chamber, the treatment, and the results have been given in detail in the report of the cases.

In both cases absence of Hansen's bacilli in the aqueous humor of an iritis with hypopyon confirmed the case report made by me before the French Ophthalmological Society. It would seem that, in leprous iritis with hypopyon, the aqueous humor does not contain any bacilli of Hansen. As for the case of Voisin and Lombard, it would seem that Hansen's bacilli were found in the aqueous by chance and could be due to their scattering from the cornea in a severely ill patient. The picture produced by the cells in the aqueous of the French case was quite different from mine because numerous red cells and polymorphonuclear leukocytes were found, while in my cases the red cells were missing and the polymorphonuclear leukocytes were rare. The specimens from the aqueous cultured on Loewenstein medium for acid-resistant bacilli were negative through all the examinations. Finally, the inoculation of some of the aqueous in the anterior chamber of a rabbit did not provoke any disease and only a slight reaction which disappeared within a week.

In my first case, the hypopyon had disappeared in 24 hours after a subconjunctival injection and, in the second case, the more severe one, the hypopyon had completely disappeared after the second injection of hy-

drocortisone. Treatment with hydrocortisone acetate (one percent) eyedrops and subconjunctival injections of hydrocortisone twice every week were continued during the first month and once every week during the second.

The appearance of a large excavation in the cornea near the limbus after subconjunctival injection of a quantity of hydrocortisone (30 and 25 mg.) near the limbus should be noted (case 2). Evidently there was a disturbance of the fluid elements in the cornea, that is, a temporary dehydration caused from the great quantity of hydrocortisone injected near the limbus. The condition did not affect the favorable influence of the drug on the course of the disease and did not produce any significant ocular signs and, finally, did not leave any permanent alterations. The conjunctival nodules did not disappear for more than three months, having a tendency to absorb slowly.

SUMMARY

Two cases of leprous iritis with hypopyon have been described. The form of the hypopyon in these cases was quite different from other hypopyon formation, being fluid and white. The examination of the albumin and cells of the aqueous humor obtained by puncture of the anterior chamber, and studied by the method of Amsler and Verrey, showed Pandy's reaction to be at first intensively positive and then to decrease progressively as the diseased condition improved. At the beginning of the disease, the number of cells per cmm. in the two cases were 224 and 486. They continued to decrease and disappeared about two months later. The morphologic picture of the cells was characteristic for leprous iritis with hypopyon during all the stages of the disease. There was always a predomination of lymphocytes and lymphocytoides. The number of reticuloendothelial system cells and iris cells and polymorphonuclear leukocytes were small. No bacilli of Hansen were found in either case.

Finally, the therapy with hydrocortisone locally gave spectacular results. In the first case the hypopyon had completely disappeared 24 hours after the first subconjunctival injection of hydrocortisone and in the second case in four days after the second in-

jection of hydrocortisone was given. Treatment with hydrocortisone locally was continued for a long period. There were no relapses.

14, Mistriotou Street (8).

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OPHTHALMIC MINIATURE

Long experience has shown that people who have had the misfortune of losing one eye have always seen more distinctly, and in a more acute manner, than they did before with both.

That this is agreeable to true philosophy has been proved by some curious observations of a learned German, Oepinus, who, in looking through a hole made in a plate of metal, about the tenth of a line in diameter, with his left eye, found that the hole itself appeared larger, and also that the field of view seen through it was more extended whenever he shut his right eye, and both these effects were most remarkable when that eye was covered with his right hand. This he very judiciously considered as depending upon the enlargement of the pupil of one eye when the other is closed, and which he considered as wisely appointed by a benevolent Providence, in order that, when one eye fails, the field of view in the other may be extended.

H. Colburn, London, 1816,

The Arts of Preserving the Sight Unimpaired to an Extreme Old Age.

NOTES, CASES, INSTRUMENTS

A TEACHING DEVICE FOR GONIOSCOPY*

ROBERT A. MOSES, M.D.
Saint Louis, Missouri

The teaching of gonioscopy has been simplified by the use of eye hemispheres mounted in celloidin blocks. These are obtained after the routine histologic sections have been taken and are mounted with cut surface against one wall of a small rectangular jar filled with thick gum damar. We made our jars from pieces of microscope slides cemented together with "Pliobond" cement. The mount is placed on a small platform attached to the chinrest of the slitlamp microscope.

If in preparation of the eye for embedding only one calotte enters the anterior chamber,

*From the Department of Ophthalmology, Washington University School of Medicine and Oscar Johnson Institute. This investigation was supported in part by a research grant, B-621, from the National Institute of Neurological Diseases and Blindness of the National Institutes of Health, Public Health Service.



Fig. 2 (Moses). Cross section and gonioscopic view of specimen seen simultaneously through biomicroscope. Note peripheral anterior synechias.

the mounted block presents an intact chamber angle. At the same time the familiar meridional section of the globe is represented by the cut edge of the eyeball. Thus, one may

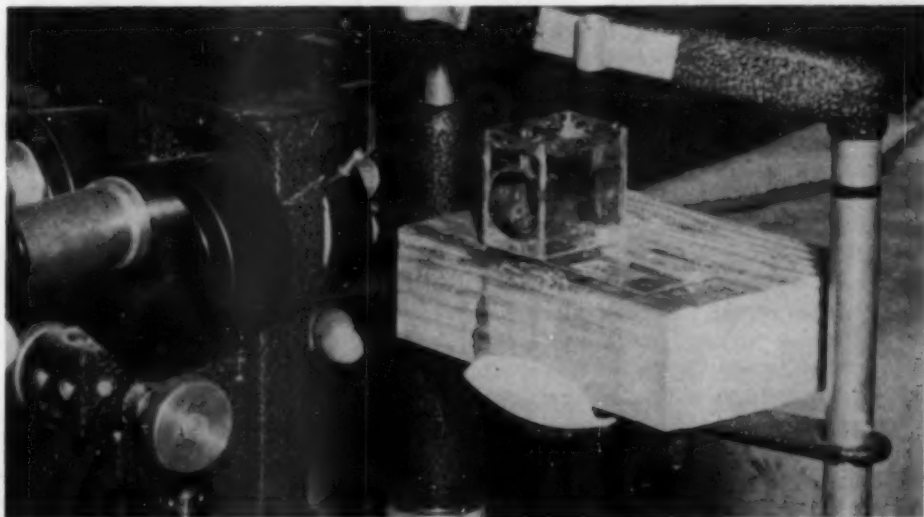


Fig. 1 (Moses). Jar containing specimen in celloidin block supported on chinrest of slitlamp.

Finally, the therapy with hydrocortisone locally gave spectacular results. In the first case the hypopyon had completely disappeared 24 hours after the first subconjunctival injection of hydrocortisone and in the second case in four days after the second in-

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If in preparation of the eye for embedding only one calotte enters the anterior chamber,

*From the Department of Ophthalmology, Washington University School of Medicine and Oscar Johnson Institute. This investigation was supported in part by a research grant, B-621, from the National Institute of Neurological Diseases and Blindness of the National Institutes of Health, Public Health Service.



Fig. 2 (Moses). Cross section and gonioscopic view of specimen seen simultaneously through biomicroscope. Note peripheral anterior synechias.

the mounted block presents an intact chamber angle. At the same time the familiar meridional section of the globe is represented by the cut edge of the eyeball. Thus, one may

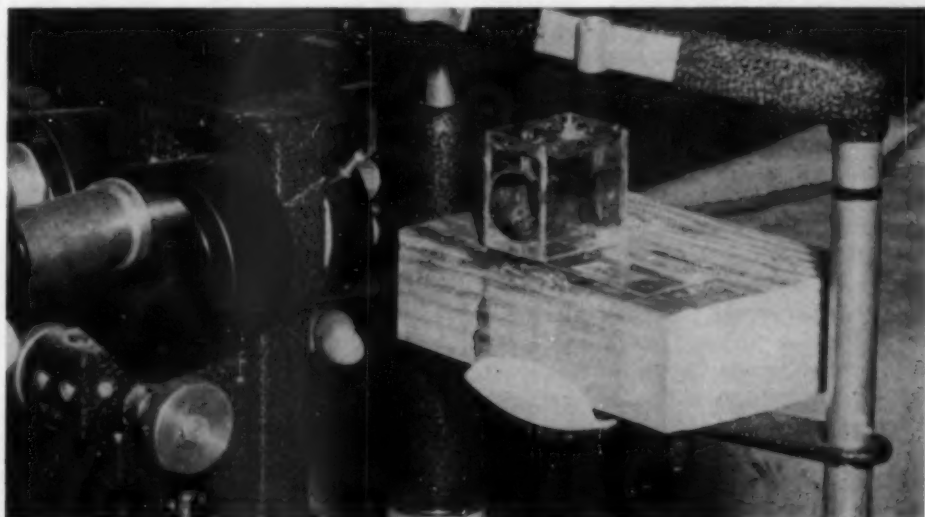


Fig. 1 (Moses). Jar containing specimen in celloidin block supported on chinrest of slitlamp.

locate a structure in the section and follow around the angle to obtain the gonioscopic view of the same structure. The appearance of the structures in the mounted specimen is somewhat too translucent and Schwalbe's line is not seen, but the device has proved useful none the less. Other anatomic features may be demonstrated in the same preparation.

No claim of originality is made in the use of celloidin blocks for demonstration of anatomy. This is an old and obvious device. It is hoped only to emphasize that these blocks are simple and valuable preparations for teaching of a particular clinical technique.

640 South Kingshighway (10).

LONG-STANDING MELANOMA OF THE BULBAR CONJUNCTIVA

EUGENE M. BLAKE, M.D. AND
ROCKO M. FASANELLA, M.D.
New Haven, Connecticut

The patient, whose condition is described, was first seen by one of us (E. M. B.) in December, 1932, for a routine examination of the eyes. The patient was at that time 63 years of age and in good health. The ocular examination revealed nothing unusual except for a narrow band of brown pigment in the conjunctiva at the corneal margin on the temporal side of the right globe.

When next seen, in March, 1955, the ring of pigment had increased in area and was slightly elevated. The biomicroscope gave the impression that the cornea was infiltrated by the tumor cells but this proved later to be due to the overlapping for 2.0 or 3.0 mm. of the cornea by the pigmented mass, thus preventing a clear inspection of the underlying tissue. Owing to the age of the patient, now 85 years of age, and the freedom from symptoms from the growth, it seemed unwise to remove the melanomatous mass. However, by December, 1956, there was a definite increase in the tumor which protruded for three mm. between the eyelids,

and was irritating and unsightly. The patient was then admitted to the Grace-New Haven Community Hospital for surgery.

Physical examination at the hospital disclosed an 87-year-old woman in excellent health. There was moderate congestion of the right globe with normal pupillary reaction. There were no skin lesions and no adenopathy was demonstrable. The breasts were negative for masses, lungs clear, heart normal and not enlarged. Pelvic and rectal examinations were negative.

A tracer dose of P^{32} of 200 cc. was given 24 hours before checking, and localization of P^{32} was done with a Geiger-Mueller counter probe over the tumor and the concentration was found to be 150 percent greater than in the control areas. Review of the skeletal system as a part of the metabolic series revealed no evidence of osteous metastases. X-ray films showed atheromatous plaques in the aortic arch and abdominal aorta. Urine and blood studies were all within normal limits. No melanin appeared in the urine.

Operation. Under local and subconjunctival anesthesia of Xylocain (two percent) the growth was carefully excised from the globe leaving a good margin of clear conjunctiva attached to the mass. The wound was closed with plain No. 6 catgut. Healing was uneventful. With the slitlamp, there were no pigment spots remaining. The corrected vision was 20/20.

A portion of the tumor was implanted into the anterior chamber of a mouse by Dr. Harry S. N. Green. There has been no growth after six months.

It is now six months since removal of the tumor and there is no evidence of recurrence.

The pathologic report by Dr. David Freeman, pathologist for the Section of Ophthalmology, follows:

Microscopic. The tissue, measuring about 12 by 6.0 mm. on section, is a malignant melanoma arising in the conjunctiva at the limbus. The cells vary in appearance in different areas, in one of which they consist of

a large ovoid, darkly staining nucleus surrounded by an abundant cytoplasm. There is a large nucleolus and many mitotic figures. In another area the protoplasm stains minimally, leaving a large nucleus centrally placed in an areolalike space. The cells are arranged in dense sheets and contain a small vessel with a sparse connective tissue mantle. A large amount of pigment is seen throughout, especially subepithelially where a bone corpusclelike configuration is present. The tumor cells do not invade the epithelial covering. No uveal tissue is seen.

Diagnosis. Malignant melanoma of conjunctiva.

Dr. A. B. Reese graciously examined the sections and reported as follows:

"I have examined the sections in the case of the elderly patient with the pigmented tumor of the conjunctiva. Certainly this is a malignant melanoma and I would subdivide it into the cancerous melanosis type. I should think in a patient aged 87 years that local excision would be adequate, for I am sure that the chances are that she will die of natural causes before the tumor would prove lethal. The tumor certainly has a very malignant cytology and were this in a younger person, then I think an exenteration would have to be done."

There are several reasons for presenting the record of this patient, (1) the known duration of the tumor for 35 years, (2) the proved malignancy, (3) the lack of demonstrable metastases, and (4) no sign of local recurrence after six months.

303 Whitney Avenue.

EMERGENCY TREATMENT OF VITREOUS BULGE AND WOUND GAPING

COMPLICATING CATARACT SURGERY

M. S. OSHER, M.D.
Cincinnati, Ohio

During cataract surgery, with adequate akinesia and retrobulbar anesthesia, one usu-

ally encounters a soft eye with an uneventful lens extraction. The complications of either bulging vitreous at the wound or less often vitreous loss at the wound are prevented by adequate akinesia and retrobulbar injection. In fact it is so important that it is well worth the extra time involved to repeat the akinesia, if the akinesia is not working in a complete manner before actually starting the operation. Either the O'Brien or the Van Lint method may be used.

Despite these precautions, occasionally an akinesia injection will wear off before surgery is completed and the patient will involuntarily or voluntarily squeeze his eyelids. This sometimes may be noted by a warning signal of twitching or spasmodic contraction of the eyelids, or by twitching of the globe and surrounding tissues themselves. In some of these eyes, once the corneal section has been completed, the base of the iris will bulge to varying degrees. If the vitreous bulge, which is the cause of the iris bulge, increases in a matter of seconds, the hyaloid membrane may rupture and vitreous escape.

This situation is a real emergency, especially before the hyaloid ruptures in cases where the bulge continues to increase, and demands heroic and immediate action. Out of sheer desperation in one such instance, the surgeon placed the ball of his index finger over the cornea and applied gentle but sufficient pressure to push easily the corneal edge of the wound, which was gaping more each second, back into place and prevent vitreous loss. While holding the wound edges closed, with the help of an assistant, preplaced corneal-scleral sutures may be tied, or if the Kuhnt type of fornix-based sliding conjunctival flap has been prepared, the flap may be pulled down over the wound and snugly tied while holding the wound edges together. Since I have not heard or read about this emergency type of procedure, namely holding the cornea in place and the wound closed with the ball of the finger, it was felt the following case history might be interesting.

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CASE REPORT

Mrs. L. S., aged 53 years, was hospitalized with bilateral cataracts for cataract extraction of the left eye. After routine preoperative sedation of Demerol, Nembutal, Thorazine, and local drops consisting of cocaine, adrenalin, atropine, neosynephrine, and argyrol, preauricular O'Brien type akinesia with 10 cc. of two-percent Novocaine was induced. One-half cubic cc. of Wydase-Novocaine solution was injected through the lid into the region of the superior rectus and 1.5 cc. of the solution remaining in the syringe was injected retrobulbarly.

Pressure over the eye was applied for five minutes. In another five minutes there was adequate akinesia and retrobulbar anesthesia. The affected eyelids could not be voluntarily closed and, unless manually closed, remained in a partially opened or lag ophthalmic position. Considering this a satisfactory preparation for routine cataract extraction, the operation was started.

A fornix-based Kuhnt type of sliding conjunctival flap was prepared, with mattress sutures through the nasal and lateral edges of the flap, respectively. A good bite of episcleral tissue was obtained at the 4-o'clock position for one suture and at the 8-o'clock position for the other suture. On testing, the flap was pulled down over the cornea snugly and covered all of the contemplated corneoscleral wound area. A superior rectus fixation suture was then inserted and a half-thickness cut was made in the corneoscleral junction at the 12-o'clock position with a Bard-Parker knife. No. 6-0 silk on an atraumatic needle was inserted through this corneoscleral area at the 12-o'clock position, and the loop bridging the cut was pulled out and laid aside so that it would not interfere with the corneal section.

The cornea was sectioned with keratome and scissors and a peripheral iridectomy done. The anterior lens capsule was grasped inferiorly with an Arruga forceps and the lens rocked a bit. Counter pressure was made

at the lower limbus and, by combined traction and counter pressure, the lens was tumbled through the wound in a routine manner without complications. The 12-o'clock corneoscleral suture was tied with the wound in good approximation.

Up to this point the eye appeared soft, the patient relaxed, and there were no signs of squeezing or extraocular muscle contracture. But, as the conjunctival flap sutures were picked up, in order to pull the flap down for tying, the globe and surrounding tissues as well as the eyelids began to twitch. The patient was squeezing. Within a few seconds the wound began to gape and the iris, pushed from behind by intact vitreous, began to bulge. The 12-o'clock stitch snapped with the knot remaining hinged to the scleral edge of the wound. A quick completion of the peripheral iridectomy to a complete iridectomy was done.

Attempted coverage of the wound with the flap could not be done speedily enough. The wound continued to gape wider by the second. The pressure was so great that the edge of the conjunctival flap, which was repeatedly pulled down over the corneal edge of the section, continued to slip back and get caught beneath the corneal lip of the wound. In desperation, and with only a matter of fractions of seconds before the hyaloid would rupture with vitreous loss, the ball of the forefinger was pressed firmly over the cornea. The wound edges, along with the vitreous and iris bulge, were pushed back into place.

Then without rushing, and with the help of the assistant, the conjunctival flap was pulled down over the wound and tied snugly while the finger maintained adequate pressure to prevent any further gaping of the wound. For extra snug covering effect and protection, since there was no longer any corneoscleral suture, two more sutures were placed from the middle of the edge of the flap to the loose conjunctival tissue midway between the lower fornix and limbus. The sutures acted as guy ropes and held the flap

and, it was hoped, the wound edges tightly. Neosporin ointment and binocular patches were applied.

In view of the fact that my last sight of the entire corneal wound under direct observation showed a gaping bulging wound, I was not certain whether the flap held the edges in good approximation or whether there was vitreous face and perhaps iris still between the wound edges. It was felt that if vitreous face bulged between the wound edges, healing would not take place and further complications would ensue. Consequently the patient was again taken to the operating room about 12 hours later and, under general anesthesia plus local akinesia, the wound edges were examined.

This necessitated cutting loose the conjunctival flap sutures in order to pull the flap up to look at the wound. Preplaced conjunctival flap sutures were inserted in identical fashion to the original ones. The wound edges were in excellent approximation. After again covering the wound snugly with the conjunctival flap, Neosporin ointment and binocular patches were applied. The guy rope sutures across the cornea were cut loose in about 48 hours and the patient was sitting on the side of the bed in a little less than four days postoperatively, with a perfectly clear cornea and formed anterior chamber. The eventually corrected visual acuity was 20/20.

CONCLUSION

This case is presented only to call attention to the possibility of a new method, not previously known to me, to handle the emergency situation of bulging through the wound in cataract extraction. Such a situation can almost always be prevented to begin with, by adequate akinesia, retrobulbar injection, and global pressure, but an occasional case will slip by with adequate akinesia at the start of the operation but with inadequate akinesia before the wound is closed.

606 Doctors Building (2).

LONG-TERM CURE OF RETINOBLASTOMA WITH X RAYS*

GILBERT W. CLEASBY, M.D.

San Francisco, California

Because of the current interest in the treatment of retinoblastoma by various methods of irradiation, this case of bilateral retinoblastoma, in which the second eye was treated with contact X-ray therapy and has been followed for 12 years, is presented.

CASE REPORT

On January 18, 1945, a three-year-old white girl was referred to the Stanford University Hospitals Eye Clinic with a history of having had the right eye removed in June, 1944, for retinoblastoma. A tumor was suspected in the left eye. Examination under general anesthesia revealed a large retinoblastoma in the inferior nasal periphery. The area of the tumor was treated by contact X rays with the following factors: 25 kv., 2 ma., 1.2 mm. Al filter, 5.0 cm. A.S.D., 1-cm. cone, 1,000 r per treatment on five successive days to a total of 5,000 r.

Periodic fundus examinations thereafter showed regression and atrophy of the tumor with formation of dustlike vitreous opacities overlying the area of involvement.

In April, 1947, the patient was found to have a 1.0 mm. by 3.0 mm. fluffy white mass of what appeared to be tumor tissue in the inferior nasal periphery of the anterior chamber. This had been noticed one week previously by the parents.

Examination revealed some increased elevation of the mass in the inferior nasal periphery of the fundus and a slight forward displacement of the iris in that area. Contact X-ray therapy totalling 6,452 r to the inferior nasal quadrant was given in five divided doses on successive days with the same factors as in the first course. At the

* From the Division of Ophthalmology, Department of Surgery, Stanford University School of Medicine.

termination of this treatment the mass in the anterior chamber had disappeared.

Five months later the tumor was found to have receded markedly and appeared quiescent. In the superior periphery were scattered flat whitish striate areas, possibly representing retinopathy secondary to the irradiation. There was some erythema of the lid margins with loss of lashes and slight hyperemia of the bulbar conjunctiva.

An incipient posterior subcapsular cataract was noted in March, 1949, with reduction of vision from the previous 20/25 to 20/30. The cataract progressed rapidly and an intracapsular extraction was performed February 18, 1950, without apparent complications. Numerous adherent ciliary processes were stripped from the lens during delivery, but the vitreous face remained intact. By May, 1950, the pupil had drawn up so that fundus examination was not possible. At this time there was considerable atrophy, epilation, and telangiectasis of the lids with dilated vessels present in the lower bulbar conjunctiva. The lacrimal passages were not obstructed and irrigated easily.

A horizontal dissection through the iris was performed June 7, 1950, and a good pupillary opening obtained. An aphakic correction provided vision of 10/20 and J1. Subsequently vision has continued to improve to the present level of 20/25 plus. There has been no further change in the appearance of the fundus, where there is a flat, white area of atrophy at the tumor site.

A small area of corneal opacification and vascularization has developed at the site of X-ray application. Trichiasis and entropion of the upper lid were successfully corrected by a free mucous membrane graft performed December 14, 1955.

SUMMARY

This case of retinoblastoma, treated with 5,000 r of contact X rays initially and with 6,452 r two years later, illustrates several of the complications of this form of therapy.

However, the patient appears to be cured and has retained excellent vision.

Clay and Webster Streets (15).

ACKNOWLEDGMENT

I wish to thank Dr. R. R. Newell of the Department of Radiobiology, Stanford University School of Medicine, for his assistance with this paper.

SYMPATHETIC OPHTHALMIA*

CONTROLLED BY CONTINUOUS CORTISONE THERAPY OVER A FOUR-YEAR PERIOD

JOHN S. CRAWFORD, M.D.
Toronto, Ontario

Up to the present time no standard therapy for the treatment of sympathetic ophthalmia has been outlined. A satisfactory method of treatment will only be arrived at when the results and the dosages of the hormone therapy have been studied in a large series of cases. It is generally agreed that relapses may occur if therapy is withdrawn too soon. The results of a number of cases of sympathetic ophthalmia treated with hormone therapy reported in the recent literature show varied results, but Haik¹ and his associates state that "relapses seem likely to occur if therapy is withdrawn, or if the dose is decreased too soon after treatment has been instituted, though the optimum dosage and duration of treatment remain to be determined." It is hoped that the clinical course of this case, as well as others presented in the literature, will help us to arrive at a more standardized method of treating sympathetic ophthalmia.

The following is a case of sympathetic ophthalmia (verified by pathologic examination) having a number of relapses as a result of withdrawal of the hormone therapy apparently too early. The inflammatory reactions were controlled by starting the treatment again after each lapse. The cortisone

* From the Department of Ophthalmology, University of Toronto, and Hospital for Sick Children.

was only discontinued after four and a half years of treatment.

This case is of clinical interest because:

1. The injury initiating the sympathetic ophthalmia was entirely corneal.

2. The sympathizing eye showed acute inflammatory signs on three occasions and each time this was controlled by hormone therapy.

3. The results of surgery to control the secondary glaucoma, even though the eye was acutely inflamed and the iris vascularized, were uncomplicated and good. This was considered to be due to the hormone therapy being given.

CASE HISTORY

On August 12, 1951, a nine-year-old boy was admitted to the Hospital for Sick Children with a horizontal laceration to the lower portion of his right cornea. The laceration measured 5.0 mm. in length but did not involve the limbus. The injury had been sustained on the evening of August 11, 1951, when the boy was stripping some bark from a tree with a paring knife which slipped and struck his right eye. The edges of the wound appeared to be opposed and there was a moderate amount of blood in the anterior chamber. The lower border of the pupil was drawn downward and appeared to be incarcerated in the wound, but was not prolapsed through the wound. The anterior chamber was of normal depth and a good red reflex was present although the fundus details were hazy due to the blood in the anterior chamber. The vision of the right eye was limited to counting fingers at 18 inches and the vision of the left eye was 20/20. The patient was put to bed and 0.25 percent eserine ointment was instilled in the right eye. Both eyes were covered.

On the second and subsequent days atropine ointment was used. On August 19th a small portion of the iris was first noticed to be protruding from the wound and on the following day, under general anesthesia, the

prolapsed iris was excised. As this was done the anterior chamber suddenly collapsed and several black silk sutures were placed in the cornea to close the wound. On August 25, 1951, the anterior chamber was deep and clear and the fundus could be well visualized. The patient was discharged from the hospital on August 26th.

The case was followed at my office and by the middle of September the eye was completely quiet and the vision in this eye had improved to 20/40. On September 20th, a slight degree of photophobia developed in the other eye and a small number of cells were seen in the anterior chamber. The condition remained about the same for the next four days. The boy was therefore admitted to the hospital and the eye enucleated on September 24, 1951. On October 5th, the patient was discharged from the hospital.

The remaining eye was quiet and remained this way until October 20th when the boy complained of some photophobia of his left eye. The left bulbar conjunctiva was injected. The vision in this eye was 20/20-2. There was no sign of keratic precipitates and no cells in the anterior chamber. On refraction, the left eye was +0.75D. cyl. ax. 75° equals 20/15 - 2.

The boy was again seen on October 26, 1951. He showed photophobia and a number of new small blood vessels were present on the iris. Ciliary injection was present and some cells were seen in the anterior chamber. The patient was again admitted to the hospital and given ACTH, 25 mg. in 500 cc. of five-percent glucose and distilled water, over a 10-hour period each day. This was continued until November 2nd.

On November 3rd the treatment was changed to oral cortisone, 25 mg. twice a day, along with the instillation of cortisone ophthalmic ointment to the conjunctival sac every four hours. By November 8th the eye was completely quiet with no cells in the anterior chamber and the vision was 20/20. The patient was discharged and continued with the

cortisone eye drops every three hours and 25 mg. of cortisone by mouth twice a day. The pupil was kept dilated with one percent atropine solution, one drop twice a day. Cortisone was continued until January 24, 1952. The eye was found to be remaining quiet and the vision was 20/15. All medications were therefore discontinued.

The child was seen at regular intervals and the eye remained quiet until March 4, 1952. At the time of examination, the pupil would not dilate well with atropine. The vision in this eye was 20/30 and was not improved with lenses. The vitreous was hazy. There were some cells in the anterior chamber but no keratic precipitates were present. Externally the eye was white with no ciliary injection present. The boy commented that he was having difficulty recognizing red objects. The condition remained the same until March 12th when he was again admitted to hospital. The number of cells in the anterior chamber had increased in number and some pigment deposits were present on the anterior and posterior surface of the lens. The vitreous was hazy. The vision in this eye was 20/50 on that day.

The boy was given 30 mg. of ACTH in 500 cc. of five-percent glucose solution intravenously daily. This was continued for 10 days. He was also placed on local cortisone ointment and one-percent atropine drops, three times a day. By March 25th the vision of the left eye had improved to 20/30. There were fewer cells in the anterior chamber and the vitreous was clear. The patient was discharged from the hospital and was advised to use cortisone ophthalmic solution, two drops in the left eye, five times a day, and one-percent atropine solution, twice a day. He was followed at weekly intervals and on May 31, 1952, a refraction was done on the left eye. The vision in this eye was 20/40 and with a $-0.5D.$ sph. with a $-0.25D.$ cyl. ax. 165° equals 20/25-2.

The only treatment carried out at home was the continued use of cortisone drops four times daily. On October 23, 1952, the

refraction was repeated and it was found that under cycloplegic the left eye took a $-1.5D.$ sph. equals 20/20-2. On fundus examination it was noted that there was cupping of the left disc. The vitreous was slightly cloudy and the tension in this eye was 48 mm. Hg (Schiötz) using a 7.5 gm. weight. The visual fields were done and found to be normal using a 1.5/1,000 white test object.

Four-percent pilocarpine was then used in the eye, four times a day. This was tried only after atropine solution had been found not to lower the tension. On October 31st, electrotonography was done and it was found that the rate of outflow was sluggish and was not increased with the use of miotics. On November 8th it was decided that some type of surgical interference would have to be carried out and a goniotomy was done on the left eye, which was followed by some bleeding into the anterior chamber.

Following the goniotomy the tension remained normal on the following day, but on the second postoperative day the tension had again risen to 45 mm. Hg and so on November 11, 1952, the anterior chamber was opened with a keratome under a conjunctival flap. The iris was grasped with iris forceps and then cut, and, after a small portion of it was torn off at the root, it was placed in the wound. A small piece of sclera was also removed. A good filtering bleb was obtained and following this procedure the tension was normalized. The patient was discharged from hospital on November 22, 1952, on a low salt diet, one gm. of potassium chloride per day, 100 mg. of cortisone per day, and cortisone ophthalmic ointment to the eye every four hours. The patient was advised to continue with the cortisone, 100 mg. a day, for two weeks and then reduce the dose to 50 mg. a day in divided doses.

The patient was seen at monthly intervals. His vision improved to 20/40 with correction and on June 10, 1955, the vision of the left eye with a $-1.0D.$ sph. $\ominus -0.75D.$ cyl. ax. 90° equalled 20/30. The vision has never been corrected better than 20/30 due to the num-

ber of pigment deposits on the anterior surface of the lens. The vision has remained at 20/30 with correction, but the boy has been continued on cortisone therapy, 12.5 mg. twice daily, up to the present time.

SUMMARY AND CONCLUSION

A nine-year-old boy developed sympathetic ophthalmia following a perforating injury to the central portion of the cornea on August 11, 1951. On August 19, 1951, the iris prolapsed through the wound necessitating excision of the prolapsed iris and suturing of the cornea. On September 20, 1951, uveitis (sympathetic ophthalmia) developed in the second eye and on September 24, 1951, the injured eye was enucleated.

The second eye settled down and remained quiet until October 20, 1951, when uveitis again developed. This was controlled by ACTH and cortisone by mouth and locally, which was continued until January 24, 1952, when, as the eye was remaining quiet, the

administration of the drugs was stopped.

On March 4, 1952, the uveitis recurred, the same treatment was reinstituted and by March 25, 1952, the eye was again quiet. On October 23, 1952, secondary glaucoma developed and, on November 8, 1952, goniotomy was done with poor results. On November 11, 1952, an iridencleisis with sclerectomy normalized the tension.

Cortisone both by mouth and locally has been continued up to the present time without further relapses.

It is difficult to decide when it is safe to discontinue the hormone therapy in cases of sympathetic ophthalmia, but with the number of relapses in this case the cortisone was not stopped until June, 1956.

Only after a number of cases have been studied will we be able to arrive at the dosage and duration of treatment required in these cases.

Banting Institute (5).

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PLASTIC SPHERES IN CLIP-ON FRAMES*

CONRAD BERENS, M.D.

New York

AND

B. EVELYN TAYLOR

Glen Cove, Long Island

An increasing awareness of the need for clip-on spheres that are lighter than glass, to be used in the management of certain eye problems, has led to the development of plastic lenses† for his purpose.

Description. These spherical lenses are made of clear, thermoplastic material 40 mm.

and 44 mm. in diameter. They are light in weight, resistant to breakage or chipping, and small scratches may be eliminated by polishing. The plus and minus plastic spheres are available in the following dioptric strengths: -0.5, -1.0, -1.5, -2.0, -2.5, -3.0, and +0.5, +1.0, +1.5, +2.0, +2.5, and +3.0. The set consists of 24 toric lenses and binocular and monocular clip-on frames (fig. 1).

Indications for use of plastic spheres.

Plastic spheres in clip-on frames are practical and especially useful in the management of accommodative esophoria and esotropia, where fairly frequent lens changes may be necessary during the training in dissociation of accommodation and convergence. They may be used for trial temporarily before being incorporated into a prescription,

* Aided by a grant from The Ophthalmological Foundation, Inc., and the Department of Research, New York Association for the Blind.

† Made by R. O. Gulden, Philadelphia 20, Pennsylvania.

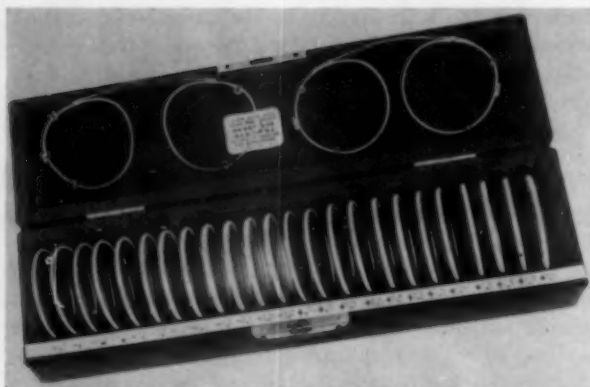


Fig. 1 (Berens and Taylor). A set of 24 toric plastic lenses, ranging from -0.5 to $-3.0D$, and $+0.5$ to $+3.0D$, with binocular and monocular clip-on frames. (Monocular frame not shown.)

where difficulty may be experienced in prescribing the most suitable correction for the patient's comfort. The plastic spheres may also be used for temporary relief of blurred vision associated with the use of cycloplegics. Plastic spheres in clip-on frames can facilitate the measurement of squint cases, particularly those with an accommodative factor. They may be used in clip-on frames in place of bifocals, by some patients.

Plastic spheres have been used in clip-on frames for over two years and they have been found to be extremely practical for home as well as for office use. The plastic spheres, as well as the plastic prisms¹ have proven practical and valuable in the management of many eye problems.

708 Park Avenue (21).
13 Walnut Drive.

REFERENCES

1. Berens, C.: Light clip-on plastic prisms for the temporary correction of heterophoria and heterotropia. *Tr. Am. Ophth. Soc.*, **52**:461, 1954.

OPHTHALMIC MINIATURE

Tho' M. Daviel gives this Operation (cataract extraction) great encomiums, yet I cannot find it answers this expectation, in a notable degree elsewhere. In London it has not met with great success, tho' in Dublin, it bids fair for coming into Repute; for my ingenuous correspondent, and learned Friend, Doctor Rutty informs me, that it has been practised with success, by Messieurs Dillon and Roony, Surgeons of Repute and experience in Dublin; but adds, that tho' each of their Patients, recovered Sight sufficient to direct them, yet the cornea was a good deal contused, and an Opacity remained on each of them.

Silvester O'Halloran of Limerick, 1750.

OPHTHALMIC RESEARCH

EDITED BY FRANK W. NEWELL, M.D.

Abstracts of papers to be presented before the Southern Section of the Association for Research in Ophthalmology, Miami Beach, Florida, November 12, 1957
A. E. MEISENBACH, M.D., *Section Secretary*

Anaerobic *Actinomyces bovis* corneal ulcer. Wendell D. Gingrich and Mary E. Pinkerton, University of Texas Medical School, Galveston, Texas.

It is rare that an anaerobic actinomycete is isolated in culture and specifically identified as in this case: Two weeks after an oyster-shell injury to the right eye, a 53-year-old white man developed a large central corneal ulcer, with considerable destruction of corneal substance. On various types of therapy, the condition progressed to descemetocoele. On culture, no bacteria and no aerobic fungi were shown but a good growth of organisms in thiolglycolate broth was identified as *Actinomyces bovis*. The organism showed some sensitivity to penicillin but little or none to other antibiotics. Daily iontophoresis with sodium sulfacetamide gave marked improvement in four days and cure of both the keratitis and iritis within 10 days.

Modern concepts in preparing the diabetic for eye surgery. B. E. Lowenstein, M.D., Metabolic Clinic, Dade County Hospital, Kendall, Florida.

Eye surgery on a diabetic necessitates that enough carbohydrate be burned to prevent ketosis and that protein balance be maintained to prevent infection and to promote tissue repair. The following program has been employed:

1. Maintenance of a moderate and sustained hyperglycemia because (a) it minimizes danger of insulin reactions and (b) it permits carbohydrate metabolism without increased insulin.

2. A diet containing at least 120 gm. protein and less than 50 gm. fat a day. As much of the carbohydrate as possible should come from fruits and honey be-

cause of their fructose content. Because the diabetic can utilize fructose without insulin, it is recommended that before, during, and after surgery, hyperglycemia be maintained by intravenous infusion of fructose.

3. Sufficient insulin to prevent ketosis but no more than this minimum.

Effects of retrociliary cyclodiathermy versus retrociliary cyclo-electrolysis of the normal rabbit eye. L. Benjamin Shepard, M.D., Medical College of Virginia, Richmond, Virginia. This study was aided by a grant from the Alfred P. Sloan Foundation, Inc.

A controlled study of 53 normal rabbits whose right eyes were subjected to retrociliary cyclodiathermy and whose left eyes to retrociliary cyclo-electrolysis showed a substantial reduction in intraocular pressure. Two comparisons are made: (1) Of the two retrociliary procedures and (2) with a previous study in which similar procedures were made directly to the ciliary body.

Prophylactic beta irradiation for the prevention of recurrence of pterygium. Seymour B. Gostin, M.D., Veterans Administration Hospital, Dallas, Texas.

An attempt has been made to prevent recurrence of pterygium after surgery by the prophylactic use of fractional dosage beta irradiation, starting at the time of surgery. A total of 200 eyes has been treated and the first 103 eyes operated and treated with irradiation resulted in a recurrence rate of only three percent. It is apparent that the dosage recommended for the Iliff applicator by another author is cataractogenic and that low dosage tends to increase the rate of recurrence.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

May 7, 1956

DR. BERNARD KRONENBERG, *President*

CORRECT FITTING OF EYEGLASSES

MR. AUREL E. MANGOLD spoke on this subject during the instruction period.

LESIONS OF OPTIC NERVE

DR. ALFRED KESTENBAUM said that two pupillary tests are available for the diagnosis of retrobulbar neuritis: Gunn's pupillary test and the pseudo-anisocoria test.

In the modified Gunn's test the eyes are alternately covered and uncovered and the pupillary movements are observed. If, for instance, uncovering of the right eye together with covering of the left eye results in a pupillary contraction, while uncovering of the left eye together with covering of the right eye results in a pupillary dilatation, a disease of the left eye causing a diminished pupillomotor effect is indicated. The pupillary movement is seen in the uncovered eye, the movement at the other eye can be seen only by peeping behind its cover.

In the pseudo-anisocoria test, the eyes are also covered and uncovered alternately; the final pupillary size, present after uncovering, is observed, rather than the pupillary movements.

The pupillary size after uncovering the right eye is compared with that after uncovering the left eye; both are measured numerically.

For example, in binocular illumination of a patient the right pupil is 3.5 mm. the left is 3.5 mm., too. In monocular illumination, the right pupil measured 5.0 mm., the left pupil 3.5 mm. This indicates a diminished pupillomotor effect of the right eye.

Only a difference of at least 0.5 mm. observable with the naked eye, can be considered as a positive sign.

Gunn's pupillary sign is more conspicuous, the pseudo-anisocoria test gives numerical results. Therefore, both tests have to be performed in every case.

Erroneously the signs may appear to be positive in normal eyes if the illumination comes from the side. Often illumination of the right eye from the right side causes a smaller pupillary size than illumination of the left eye from the right side. This difference in reaction is probably due to a greater pupillomotor effect of the nasal half of the retina, in accordance with certain observations of Hess.

Therefore, in the two tests, the illumination must be homogenous or symmetrical to the two eyes.

A positive Gunn's sign and a positive pseudo-anisocoria sign are significant not only for retrobulbar neuritis, as Gunn has assumed, but for any lesion of the optic nerve between eye and chiasma. Cases of positive signs in a traumatic lesion of the nerve and hemangioma in the orbit are reported. The signs are not available in bilateral nerve lesions such as nicotine amblyopia except the lesions of the two nerves are very different in degree.

The sensitivity of the tests is surprisingly high. In the period of recovery the signs may be still recognizable, when the vision is restored to almost 20/20. Finally, of course, the signs become negative.

In extensive fundus lesions, the signs may also be positive, but here the differentiation is easy. In small, circumscribed lesions of the fundus, even with a definite central scotoma, the signs are negative.

In retrobulbar lesions, routinely made measurements of the visual acuity in extramacular areas frequently revealed a dimin-

ished visual acuity in the entire field, a kind of general "depression" of vision in the sense of Traquair. While, for instance, at 30 degrees from the center there is normally finger-counting at four or five feet, in a retrobulbar lesion the vision may be only finger-counting at one foot.

This general damage of vision in the entire field of vision may explain the diminished pupillomotor effect in retrobulbar lesions with even a small or only relative scotoma. Summarized, the two tests are objective signs of retrobulbar lesions and allow a differentiation from functional disturbances or from faked amblyopia on the one hand and from circumscribed macular lesions (hemorrhage, degeneration, and so forth) on the other hand.

Positive signs are indicative of a retrobulbar lesion, negative signs make the presence of a retrobulbar lesion improbable.

TEMPORARY POSTCATARACT BIFOCAL LENS

DR. DEWEY KATZ said that, although various types of temporary postcatract bifocal lenses have been utilized in isolated instances by some ophthalmologists in the past, recently, because a type of bifocal lens which readily lends itself as a temporary postcatract bifocal lens has been devised, and a loan service has been established, the temporary postcatract lens has been used in innumerable instances by a rapidly increasing number of ophthalmologists.

When, for various reasons, the original postcatract prescription is to constitute the permanent lens, it is usually ordered three to four months postoperatively, with all that this delay implies as regards the limitation of the patient's activities and his mental reactions. This delay is made necessary in the hope that the postoperative refractive change has come to a standstill and the prescription, primarily because of its cost, is both the original and final one.

The temporary postcatract lens does away with all this, for the temporary postcatract lens is the tentative R/x which eliminates

the necessity for frequent regrinding or expensive replacement of whatever type permanent cataract lens is prescribed.

The temporary postcatract lens consists of an edged 44 or 46 mm. round planoconcave, cylindric lens. This permits the rotation of the axis of the cylinder to the axis desired. To this carrier cylindric lens there is cemented a 34 mm. round and edged convex spherical lens with a fused straight top bifocal segment. Cylinder power is available from $-0.25D$. to $-3.0D$. in $0.25D$. gradations. The planoconvex spherical lens is available from $+9.0D$. to $+14.0D$. in $0.5D$. gradations. The bifocal segments are $+2.75D$. The temporary postcatract lenses are relatively thin, light in weight, accurately ground, and cosmetically passable. The frames supplied are of metal with varying D.B.L.s and temple lengths.

The temporary postcatract lens service consists of supplying to the patient by the optician within a day or two after receiving the R/x the type lens just described on a temporary loan basis. These lenses are changed as often as the ophthalmologist believes necessary. If, as is usually the case, several changes are made in the postcatract period before the permanent frame and lenses are secured, the patient surrenders the pair of temporary postcatract lenses he has for the pair he is to secure.

When the permanent lens is ordered, it can be made up in any type cataract lens desired. It, however, is invariably advisable to have the same form grinding as in the temporary postcatract lens so as to reduce to a minimum the problems of vertex distance, segment height, and other factors.

A case was reviewed to demonstrate the frequency and the number of changes—five—of the temporary postcatract lens made available to the patient. Because of this the permanent postcatract lens was not prescribed for five and one-half months postoperatively and there was no subsequent change in prescription.

The second case presented demonstrated

the fact that temporary postcataract lenses can be prescribed for and worn to advantage by the patient as early as 10 days after cataract surgery.

The third case presented demonstrated the value of the temporary postcataract service in a patient who had bilateral cataract extraction. This patient received her first pair of temporary postcataract lenses eight days after discharge from the hospital, had six different pairs of temporary postcataract lenses during a period of eight months, when the permanent postcataract lenses were prescribed. These lenses have not had to be changed. The free use of the temporary postcataract lens service, whenever used, has been of immense benefit to the patient and to the ophthalmologist.

LAMELLAR UNDERMINING

DR. MAX CHAMLIN presented a new procedure which was described in an article published by him and Dr. Karl Rubner, recently deceased, in *THE AMERICAN JOURNAL OF OPHTHALMOLOGY*, issue of April, 1956, page 633.

Discussion. DR. MILTON BERLINGER congratulated Dr. Chamlin on the ingeniousness of the procedure. He predicted that during the next five years, we will probably have 50 to 75 new methods to shorten the eyeball. He hoped this happens because, like the operations for glaucoma and cataract, we have to keep on improving them.

The idea of infolding sclera in a way is not entirely new. He thought it was Dr. Thorpe who modified the procedure of Dr. Weve when he sacrificed the sclera before invaginating its entire thickness.

He said that he is still doing lamellar resections of the sclera. On several occasions he has used Dr. Schepen's polyethylene tube. The last case he did was a detachment in a young boy in whom the vitreous was completely degenerated and the retina was completely detached; he used the threaded polyethylene tube completely around the globe. When the tube was tied, the globe looked like a mushroom. To our surprise, some

months later the resident called his attention to the case to see the remarkable anatomic result that we got. There was a slight improvement in vision. The anatomic result was quite astonishing.

Personally, he is not convinced that the use of the short tubes has had any great merit. His reason for favoring the lamellar resection is not so much for the shortening effect, but for the manner of treatment—cauterization. He feels that the closer we can treat the choroid, the more advantageous the operation will be. If we could treat the choroid directly, he would prefer it even more so. When one treats a case in which a lamellar resection is done, one is amazed how little reaction occurs in the eye. He does very little cauterization besides that.

DR. CHAMLIN said that he was acquainted with Dr. Thorpe's modification of Weve's procedure, which is quite different from his procedure. Dr. Thorpe invaginated the entire thickness of the sclera, while he undermined the lamella on each side, thus allowing a thinner wall to be folded, insuring easier coaptation with less tensile rigidity, which would tend to unfold the sclera prematurely. He agreed that the cauterization is quite important, and, as he described in his presentation, he cauterized in the undermined area on either side of the curled-up lamella, very much according to the original technique of Dellaporta.

DR. ALAN H. BARNERT said that one of the troubles with previous lamellar resections is that the infolding sometimes does not last very long; the present modification might be expected to be more permanent. He asked whether any work had been done to determine whether this is so.

DR. CHAMLIN replied that work on the comparative permanence of his procedure is now in progress in our laboratories.

Jesse M. Levitt,
Recording Secretary.

YALE UNIVERSITY
CLINICAL CONFERENCE

March 9, 1956

DR. R. M. FASANELLA, *presiding*

DR. LEWIS LEVY, Department of Neurology, spoke on "neurologic complications of diabetes affecting the eye," and included optic atrophy, palsies of the 3rd, 4th, and 6th nerves, and pupillary changes resembling Argyll Robertson pupil. Dr. Levy divided these complications up into:

A. Those due to diabetic neuropathy type of pathologic process resulting from the metabolic abnormality of the condition.

B. Those complications of arteriosclerosis, which involve various cranial nerves.

He gave three brief case reports illustrative of this latter type of condition.

The first patient was a 69-year-old woman with a history first of transient 6th nerve paralysis in 1948. In 1953 the development of drooping of the right lid and pain in the right eye followed. Arteriography showed arteriosclerotic aneurysm of the carotid passing through the cavernous sinus affecting the cranial nerves, which were responsible for this condition. The condition improved spontaneously within a month.

The second case was that of a 77-year-old woman, who in 1952 noted a gradual drooping of the right upper lid and in 1953 developed pain in the right eye with palsy of the 3rd, 4th, and 6th nerves and first division of the 5th nerve. This also proved to be a carotid aneurysm.

The third case was that of a 76-year-old man, who, in 1953, noted poor vision in both eyes and noises in the head. There was blood pressure of 180/95 mm. Hg, bilateral proptosis, and fixed globes. Vision in the right eye was 2/200 and in the left bare light perception. Bilateral papilledema was noted and a bruit was present over each eye. X-ray arteriography showed a carotid aneurysm in the region of the cavernous sinus with a definite leak of the opaque material into the cavernous sinus. This was undoubtedly responsible for the bruit noted. In some cases

of diabetes, therefore, extraocular muscle palsies may be due to carotid artery abnormalities.

DR. PHILIP BONDY, Department of Medicine, stated that the chief problem today is the handling of the complications of diabetes. The treatment of the carbohydrate metabolism seems to be fairly well handled in general. At least the patients should be controlled well enough so that they do not go into acidosis and they are in a good state of nutrition and activity. There has been noted an accelerated rate of blood vessel deterioration. Earlier arteriosclerosis and arteriolosclerosis develop and are manifested in the usual ways, such as cardiac infarcts, arteriosclerosis of extremities, and so forth. In addition, special difficulties develop in diabetes in the kidneys, eyes, and possibly pancreas. Causes of the changes in the latter three conditions are mysterious. One theory is that poor control of diabetes is a reason for the accelerated degenerative changes. This is a very difficult situation to prove.

Dr. Bondy felt that the so-called "difficult patient" may possibly have a different type of diabetes and is psychiatrically different in addition. He referred briefly to the work of Wilson, Root, and Marble in a paper of May, 1951, in which the well-controlled type patients showed less calcification of the arteries and a smaller percentage of retinitis than the poorly controlled patients. However, Dr. Bondy pointed out that even in the so-called well-controlled patients, one third had degenerative changes and in the poorly controlled group, half of the patients did not have degenerative changes. Therefore, although there is undoubtedly some advantage to better control of carbohydrate metabolism, this is not the whole answer to the problem.

In further answer to the theory that the carbohydrate defect causes complications in diabetes, it was noted that first, complications are not proportional to the severity of the diabetes; second, complications may occur before the diabetes produces symptoms of the disease; and, as above noted, complications may occur in spite of good control and may not occur even in poorly controlled patients.

It is possible that a general causative agent for diabetes produces two great groups of effects:

First, the metabolic effects, including the low carbohydrate utilization, high protein destruction, high fat turnover, sodium potassium and magnesium loss, and acidosis conditions.

Second, the vascular effects which include vasa nervorum, atherosclerosis, and retinitis. The vascular effects may run in a parallel line, but independent of the metabolic effects.

Dr. Bondy felt that sharp control was not the answer to the problem and may be dangerous as well. Hypoglycemic episodes may lead to increased bleeding. In some cases emotional upsets may well be related to increased hemorrhages. He is skeptical about the usefulness of Rutin, vitamins B₁₂ and similar therapy. He briefly mentioned a recent work which showed insulin acts somewhat as an antigen and some of the conditions may possibly be of an allergic or sensitivity nature. Possibly the degenerative conditions would be related to an antigen antibody phenomenon.

In response to a question of Dr. Van Heuven who asked whether a certain duration of diabetes automatically brings retinopathy, Dr. Bondy answered in the negative saying that he did not believe this was true. He mentioned two patients under his care with 35 and 37 years' duration of diabetes respectively without any retinopathy.

Dr. VAN ECK has been interested especially in the lipids and their relation to diabetes. He notes that serum lipids are frequently elevated in diabetics, especially in poorly controlled diabetes. Diabetics usually have a diet containing much lipid. He studied the diurnal serum lipid pattern with patients on regular and extremely low-fat diets. On regular diets the diurnal pattern shows large variations in serum cholesterol and a very high level of cholesterol. On extremely low-fat diet the cholesterol levels become much lower and much more uniform throughout the day. This phenomenon was also noted in relation to neutral fats in the blood. The

first conclusion of his studies was that it was possible to reduce serum lipids markedly by a very low-fat diet.

The question was whether this means anything in terms of the development of arteriosclerosis or other complications of diabetes. It is not entirely definite whether the exudates in the retina in diabetes are lipid. However, several patients were followed on an extremely low-fat diet who had widespread exudates in the fundus.

Fundus photographs of these cases showed marked disappearance of the exudates in the course of six months to one year on a special diet. Hemorrhages were not affected. Dr. Van Eck believes diabetic retinopathy pathogenesis involves two factors; the vascular factor and another factor related to serum lipids. The vascular factor is evidently not affected by this particular diet. Incidentally, the diet is very disagreeable, not without hazard, and not possible in every patient. It is interesting that despite the extremely high carbohydrate values in this diet, the insulin requirement only goes up moderately.

Discussion. DR. VAN HEUVEN: Possibly the defects in the blood vessels are the primary ones and this may allow the blood elements and later lipid to go through the vessels. A question arises if a similar diet might not help exudates in hypertensive patients.

Dr. VAN ECK: Similarity of exudate may possibly lead to similar results, but this is purely speculative.

Dr. DESUTO NAGY: I observed one patient with nondiabetic retinitis circinate who is the wife of a dairy man and evidently on a diet containing lots of milk and in whom retinitis circinate disappeared spontaneously in the course of two years.

Dr. ROSENTHAL: In connection with microaneurysms, a small venule dilates due to lack of oxygen. This disturbs the endothelium and possibly endothelial proliferation results in formation of microaneurysms.

William I. Glass,
Recording Secretary

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EXPERIMENTAL OPHTHALMOLOGY: II

With the growth of research programs the administrators of foundations, the executives of industry, and the directors of the various federal groups which support research are faced with a challenging and difficult problem—the intelligent distribution of the funds as their disposal so as to yield the most constructive returns. The broad sup-

port given to ophthalmic studies and the rising tide of eye research indicate how well they have performed their task as it pertains to ophthalmology.

The prospective grantee seeking a source of funds for a research project would do well to consult *America's Foundations and their Fields* (American Foundations Information Service, 860 Broadway, New York). The volume describes the objectives

and programs of 4,162 foundations, indicates the fields supported by each, and contains a useful index of fields that will point out to anyone which groups might be interested in his project. The news columns of *THE AMERICAN JOURNAL OF OPHTHALMOLOGY* carry frequent reference to the grants of the various eye groups to support ophthalmic research. Additionally, the National Committee for Research in Ophthalmology and Blindness (Office of the Secretary, 950 East 59th Street, Chicago 37, Illinois) is prepared to offer advice on possible sources of financial support to the authors of research proposals. Information concerning the Department of Health, Welfare, and Education program in ophthalmology may be obtained from the Institute of Neurological Diseases and Blindness, the National Institutes of Health, Bethesda 14, Maryland.

The types of support available for research fall into several categories: project support, research training programs, and traineeships.

Project support varies considerably in the dollar amount available and the time period of the aid. Grants by the National Institutes of Health are usually made on the basis of a five-year commitment. However, each year, Congress must appropriate the funds before the grants can continue. There has never been a failure of continuity and with present federal legislation pertaining to the budget, there is no hope that the government would ever guarantee grants for periods extending after the elective terms of the legislators had expired. In the research program for the fiscal year, 1956 (*Am. J. Ophth.*, 42:307, 1956), the dollar value of each grant from the National Institute of Health for eye research varied from \$1,064 to \$24,490. A total of 110 projects was authorized in the amount of \$1,461,500. Considering the magnitude of the problems and the socio-economic costs of blindness, this is indeed a modest sum and suggests that the total amount expended on eye research annually by industry, universities, and the government must be less than five million dollars.

For the unknown and inexperienced investigator there have been several encouraging developments in recent years. The National Institute of Neurological Diseases and Blindness makes grants of up to \$2,000 without review of the study sections and for one year only. The National Society for the Prevention of Blindness and the National Council to Combat Blindness each encourages application for grants-in-aid which vary from several hundred to several thousand dollars. Many of these grants have been unusually helpful, some supplying funds for a single piece of equipment needed for a study, the remainder of the support being provided by the ingenuity, interest, resourcefulness, and do-it-yourself ability of the grantee.

The modest sums used for eye research point up the most serious problem facing administrators today—the shortage of trained investigators. Figures published by the *Journal of the American Medical Colleges* indicate that the percentage of medical school graduates entering ophthalmology has remained constant during the past several decades. However, not nearly enough trained ophthalmologists have remained in teaching and investigation to provide the manpower necessary for the development of research groups. In an attempt to interest medical students and ophthalmic residents in academic careers (since the majority of research is done in universities), the National Institutes of Health have instituted training programs, traineeships for mature scientists, and summer fellowships for medical students. The National Council to Combat Blindness also provides funds to attract medical students into ophthalmic research by grants for summer vacation studies in eye laboratories. These programs have been operating so short a time that their long-term success cannot be evaluated but already several workers have continued careers which began when they were medical students in ophthalmic laboratories. The need for these training programs is evident to anyone who visits and observes the paucity

of investigators in ophthalmic laboratories.

If half-time spent in research characterizes a research scientist, then there are probably less than one hundred such men in ophthalmic research in the country. It is most important that ophthalmologists be trained as research scientists and then, when qualified, be offered the opportunities and the financial support required so that they will remain in the laboratory and accomplish what they have been trained to do.

Frank W. Newell.

OPHTHALMIC PATHOLOGY CLUB

In April, 1957, the 11th consecutive annual meeting of the Ophthalmic Pathology Club was held. These meetings have proved so successful that the club now fills a unique niche in American ophthalmology, possibly in American medicine. It would seem appropriate at this time to record the origin, the development, and the activities of this group. Its continued success is due not only to its filling a definite void but also in doing it in a most unusual manner.

Prior to the formation of this group, there was no meeting at which pathology of the eye was the chief interest. In many eye meetings reports on various pathologic subjects were occasionally presented but there was no opportunity for the detailed study of ophthalmic pathology by a group particularly interested in that subject. During a discussion of this point at the Wilmer Residents Meeting in the spring of 1945, it was decided to contact a number of ophthalmic pathologists to explore the possibility of forming an organization whose primary objective would be the study of eye pathology. A group of 20 prospective members was invited to attend an organizational meeting the next October at the Academy meeting in Chicago. Practically all 20 appeared at the meeting and were most enthusiastic about the idea. The group at that time was increased to 30, at which number the membership has been consistently held. At the organizational meeting, tentative decisions were

made on the time and place of meeting, organization of the group, and the type of program. It is most interesting that these decisions have remained essentially unchanged.

The first meeting was held the two days before the Wilmer Residents Association meeting in April, at the Army Institute of Pathology, now the Armed Forces Institute of Pathology in Washington. With the exception of the meeting in 1953, which was held in Philadelphia at Wills Hospital, all meetings of the Ophthalmic Pathology Club have been held at the Armed Forces Institute of Pathology in early April. The A.F.I.P. has proved an ideal meeting place not only because of the facilities for microscopy and its excellent projection equipment but also because of its large store of interesting slide material. Selected cases from its files are reviewed from time to time during the meetings for comparison with the case being presented.

The original decision that this group should be an informal club with as little organization routine as possible has held true to the present time. There are no officers, no dues, no written constitution, no by-laws. For a number of years Dr. Rones acted as the local committee on arrangements in Washington. In recent years, there has been a "steering committee" of three members, the retiring senior member nominating the new member each year. This committee collects and lists the case reports for the program and makes arrangements for the annual dinner. There is no regular presiding officer, the members preside in rotation at the four half-day sessions. There are no dues, the total cost of the meeting being divided pro rata among the members.

On the evening of the first day's meeting, the annual dinner is held. On two such occasions, members of the club have been honored for their contributions to American ophthalmic pathology and to the success of the club—Dr. Frederick H. Verhoeff in 1951 and Dr. Benjamin Rones in 1956.

Limitation of the group has remained

constant because the program consists entirely of microscopic work and 30 members seems an ideal number. Because of necessity membership is limited, it was soon decided that individual members could not bring guests; however, if a member cannot attend he may send a proxy who will present his case and take part in the discussions. The chief of Ophthalmic Pathology at the Armed Forces Institute of Pathology is an ex-officio member.

Of the 30 original members, about 20 are still active and are usually enthusiastically present at the meetings. From time to time, as vacancies occur, new members have been added by vote of the club. Several members have retired and other members have left the club because their activities no longer include participation in ophthalmic pathology. At the present time only one member has died—one of our most distinguished and beloved members, who had been active in the group since its organization—Dr. Jonas S. Friedenwald. His absence has left a large void at the meetings of the group, as his discussions here were as outstanding as his contributions elsewhere.

Again, it is amazing that the exact form of the program originally suggested at the organizational meeting has been continued without change throughout the years. Originally, it was suggested that, as a trial, the first program should consist of case reports only. It was decided that each member should select the most interesting case coming through his laboratory the preceding year. Thirty histologic slides of this case should be furnished with an adequate protocol. These slides were to be distributed among the members for individual study at the time the member presented his case. This procedure has proved so satisfactory that it has been used at all the meetings.

The case presentations are followed by an informal discussion by the membership, not only of the diagnosis given by the member but also of details of the histologic findings and the significance of the material presented. The members often disagree and

discussions are heated, though friendly.

Each member, returns home with slides of 30 unusual cases for future study and presentation to his local group. These slide collections of the Ophthalmic Pathology Club reports have become valuable reservoirs of interesting pathologic case material. Inasmuch as these meetings have been held at the Armed Forces Institute of Pathology, these cases have been accessioned into their files for future reference at any time.

During the first few years it was of some concern to the members that the club could continue to function with the program consisting merely of interesting cases. It would seem, on first thought, that there might be a paucity of appropriate cases. However, it has become apparent that most laboratories in this country have at least one extremely worthwhile case each year so that the programs have continued at the same high level. At no time have formal papers or case reports been considered for the program.

In retrospect, the cases presented to the club seem to fall into five general types. First are those cases of interesting pathologic material not usually available for ordinary study. These cases may be common clinically but usually the disease process does not result in histologic material. The second group consists of curiosities and rarities that occur so infrequently that some individuals would probably never see them if they were not presented before such a club. The third large group includes the more unusual pathologic conditions in which there are additional findings of significance. In the fourth category are the new pathologic processes resulting from new therapeutic procedures, for example, retrolental fibroplasia, cortisone complications, scleral buckling, and so forth. Finally, there are those cases which can be classed in the "what is it" file. In these particular cases very often the membership is polled for possible diagnosis. In one instance there were three possibilities presented and the vote was 10, 10, 10.

Many of the cases presented at the club have been presented the following October

as a clinico-pathologic case report to start a session of the Eye Section of the American Academy. A number of the cases given at the club meeting have provided basic material for published papers, as well as for simple case reports.

Although the club has been flourishing for 10 years, it is not mere coincidence that there is practically nothing in the ophthalmic literature concerning the club. About the only recorded remark regarding the club was made by one of its members, Dr. Algernon B. Reese, in his presidential address before the American Academy of Ophthalmology in 1955. In this address entitled "Pathology" he states: "In the wake of all of these factors, interest in ophthalmic pathology reached a new height several years ago in the organization of the Ophthalmic Pathology Club. This group of 30 members meets once a year for two full days. Each member presents an interesting case and distributes sections to his fellow members. The discussions are stimulating and provocative."

At no time has there been any real concerted effort to collect the cases and the discussions into a sort of *Transactions* of the club, as these discussions have never been transcribed. It has always been the opinion of the members that any recording would lessen the value of the discussions because the discussor would tend to become more formal and less spontaneous in his opinions. Although many of these cases have been reported in the literature, if the member so wished, the discussions exist only in the personal notes of the members.

Therefore, in its first decade of activity the Ophthalmic Pathology Club has progressed from a puny infant of questionable origin to a healthy, active adolescent. Like most children, during this period of growth the club has been chiefly interested, perhaps selfishly, in its own activities, since the programs have been of value chiefly to the members present at the discussions. In the future there is a growing challenge for the club, not only to present this material for the enrichment of its own members, but also to

find some means to pass on this valuable store of unusual cases so that American ophthalmology as a whole may benefit.

Benjamin Rones,
John McLean,
T. E. Sanders.

CORRESPONDENCE

REMOVAL OF SCLEROCORNEAL SUTURES

Editor

American Journal of Ophthalmology:

I have read the paper by Dr. B. J. Curtin and Dr. T. L. Boyes on "The removal of sclerocorneal sutures" (*Am. J. Ophth.*, 42:421, 1956). In this paper the authors make special reference to removal of sutures after cataract extraction and point out that this problem is still much discussed. This I do not understand for, in my opinion, the problem has already been solved.

It is not necessary to use the various kinds of scissors recommended by several authors (Stallard, *Modern Trends in Ophthalmology*; Harrison, *Am. J. Ophth.*, 35:1207, 1952; Hilding, *Arch. Ophth.*, 24:371, 1940). A simpler and less dangerous way is to grasp one end of the suture with a very fine forceps which has no indentations (watchmaker's forceps) and cut out one arm of the suture with a small piece of razor blade held in a needle-holder. I still use this procedure to remove sutures after keratoplasty and other operations when the sutures produce irritation and do not fall out spontaneously.

This method need not, however, be practiced today. There are authors (Stocker, *Am. J. Ophth.*, 42:730, 1956) who prefer catgut with which I have had no experience. In my opinion this suture material is difficult to use; its knot is always thick and absorption produces strong local reactions. In addition, it is more expensive than silk.

For the past three years, I have been using silk-worm gut in cataract operations and keratoplasties (Barraquer, *Personal communication and Am. J. Ophth.*, 41:856, 1956). Silk-worm gut is produced by the silk glands of a caterpillar known as Bombyx

mori (Marden, National Geographic Magazine, 100:100, 1951).

This worm spins filaments that vary in form and size. The thickness varies from 1/2,000 of an inch to 21/1,000 of an inch. Anglers have a special interest in the thicker more resistant filaments which they use to knot salmon flies. The breaking point of the thinner filaments is about three pounds; those used by fishermen, about 17 pounds. This is the same silk used by surgeons all over the world. The filaments are woven together, the diameter depending on the type of surgery for which the sutures are intended.

The type of silk-worm gut I like best to use is the finest filament (1/1,000 to 1/2,000 inches). It is used in the same state as the worm produces it (virgin silk). It is yellowish white in color and comes in filaments that are 15 to 20 inches in length. It is thinner than the 6-0 silk or a woman's hair, which some surgeons use. Silk-worm gut may be obtained dyed blue from some manufacturers of surgical materials. Or the surgeon can dye the gut himself by dipping it in a one- or two-percent solution of methylene blue. Dyeing makes the material easier to use because it is more easily seen. It can be sterilized with instruments at 120°C. for 10 or 15 minutes without losing its strength. The blue dye disappears in a few hours and the suture can be seen with the slitlamp as a sparkling filament. Because of its extreme thinness, the suture causes no irritation to the eye. It is not absorbed but it need not be removed because it slips away spontaneously. If necessary, however, it can be removed with forceps.

I use this virgin silk for conjunctival sutures in surgery for cataracts, glaucoma, retinal detachment, strabismus, and similar procedures. It is used for the corneal edge-to-edge sutures in corneal transplantations. For cataracts, I use two sclerocorneal sutures which are covered by a small flap two to three mm. in width. The flap is sutured to the other end of the bulbar conjunctiva with four separate stitches. The sclero-

corneal sutures remain buried and, after many days, they spontaneously perforate the conjunctival flap, disappearing with no disturbance whatsoever to the patient.

These sutures are so fine that they can only be seen with a magnifying glass or the slitlamp. Sometimes, when they become imbedded, it is better to remove them with extremely fine forceps about 20 to 30 days after operation. This procedure causes no danger or inconvenience to the patient. I never remove these sutures in glaucoma or retinal detachment operations. They are especially convenient in strabismus operations, for suture removal in children is really a second operation.

(Signed) Jorge Malbran,
Buenos Aires, Argentina.

BOOK REVIEWS

DEVELOPMENTAL ABNORMALITIES OF THE EYE. By Ida Mann, M.D. Philadelphia, J. B. Lippincott Company, 1957, edition 2. 419 pages, 286 illustrations, extensive bibliography, index. Price: \$15.00.

Ida Mann's books on the embryology and developmental anomalies of the eye have become classics in the field of ophthalmologic education. It is therefore timely that the book on developmental anomalies should again have been made available to the ophthalmologist and the student through the second edition, the first edition printed 20 years ago having long been exhausted.

The book not only covers the congenital anomalies but also the developmental anomalies that occur during life. A definite distinction is made between those anomalies that are present at birth and those that occur in postnatal life. For example, in discussing dysostosis multiplex it is pointed out that the anomaly is not present at birth but appears before the age of three years; it is therefore developmental but not congenital. The book is based on a thorough knowledge of anatomy (both human and comparative), genetics, heredity, and pathology. One wishes, however, that a little more space

had been given to genetics and heredity.

While the book has been reset and the format changed to some extent, it is primarily a reprinting of the first edition having, with few exceptions, the same illustrations used in the first edition. New material has been added here and there; for example, under abnormalities of the fundus oculi a rather detailed description of retrolental fibroplasia has been added, in which the clinical entity, the microscopic pathology, and the role of oxygen as an etiologic factor are discussed. In addition, several pages have been added on persistent hyperplastic vitreous, retinal dysplasia, and the encephalophthalmic syndrome of Krause. Congenital vascular veils in the vitreous is also discussed in considerable detail and two new illustrations (fig. 133-A and fig. 133-B) have been added.

As pointed out by Dr. Berens in the introduction to the book, the advice concerning the management of patients with congenital anomalies should be read by all ophthalmologists and those interested in handicapped children. In addition, a knowledge of the subject will also help the ophthalmologist to know what to expect when surgical correction of the anomalies is attempted.

As for the book itself, while the text and illustrations for the most part are identical with the first edition, the printing of the text and the reproduction of the illustrations are superior to the preceding volume. The bibliography has been revised and brought up to date and the index has been enlarged. The format is up to the usual high standard of J. B. Lippincott Company's books.

The book is recommended for all ophthalmologists who do not possess the previous edition and it is a valuable addition to the library of those who were fortunate enough to have acquired the first edition.

Frederick C. Cordes.

TRANSACTIONS OF THE AMERICAN OPHTHALMOLOGIC SOCIETY 1956, VOLUME 54. New York, Columbia University Press, 1957. 832 pages, index. Price: \$18.00.

This volume is a collection of about 20 papers presented at the 1956 meeting of the American Ophthalmologic Society. Most of these have been or will be reprinted in *THE JOURNAL* but, unfortunately, without the discussions that so enliven the presentations. Special mention should be made of Leopold's superbly illustrated article on the effects of retrociliary diathermy on the choroidal circulation and Hogan, Thygeson, and Kimura's paper describing 38 cases of rheumatoid arthritis. Derrick Vail, in a well-controlled study, shows that Diamox is ineffective in preventing hyphema after cataract extraction. (Dr. Verhoeff's comment and Dr. Vail's response are delightful.) Birge discusses malignant glaucoma in detail and offers valuable suggestions for the recognition and treatment of this terrible complication.

Ten theses occupy the last half of this volume. These are valuable monographs. For example, Grant covers the action of non-aromatic quaternary ammonium compounds on the eye in an encyclopedic fashion, and Merriam reports in detail 100 cases of radiation cataracts. He compares this group with 73 patients who received radiation but did not develop cataracts and so is able to draw valuable conclusions as to cataractogenic doses in humans. Of particular interest is Henderson's exhaustive monograph on essential blepharospasm. He discusses all phases of the subject and points out the possible anatomic sites of the lesion in this distressing condition. Unfortunately these organic lesions are rarely seen clinically. The author rather arbitrarily separates "psychic blepharospasm" from the so-called essential type. A most interesting case can be made for the psychic etiology of all types of blepharospasm on the basis of a desire by the patient to shut out deeply suppressed unpleasant visual experiences. However, this

paper is certainly the last nonpsychiatric word on the subject.

David Shoch.

GIFFORD'S TEXTBOOK OF OPHTHALMOLOGY.

By Francis Heed Adler, M.D. Philadelphia, W. B. Saunders Company, 1957, edition 6. 499 pages, 277 figures, 27 color plates. Price: \$8.00.

When the late Sanford R. Gifford prepared the manuscript for the first edition of this widely used *Textbook of Ophthalmology* in 1938, his primary purpose was to present material which would be of value to the medical student and general practitioner rather than to the ophthalmologist and this has remained as the guiding principle through subsequent editions. In this new sixth edition, the third to appear under the authorship of Dr. Adler, there has been a conscientious effort to delete material of interest to the specialist and to add new information, such as the emergency treatment of ocular injuries, which should be available to the reader for whom the book is intended.

It is certainly most difficult to keep a textbook of this nature within the limitations necessary if it is to be used by undergraduate students of medicine whose crowded curriculum leaves ophthalmology in an all too minor role. This edition is slightly larger than the last and might have been still further improved by further condensation of certain chapters. As a reference book for students and general practitioners, however, it serves an excellent purpose in presenting everything they are likely to desire or need to know about the eye.

The author writes, as always, clearly and to the point with a minimum of ambiguity. The illustrations are numerous and excellent, selected with care from a wide variety of sources. It is obvious that the experience of previous editions and thoughtful rewriting and editing have resulted in an improved and thoroughly up-to-date book on the eye which will increase the popularity of this

work as one of the standard American texts.
William A. Mann.

THE MERCK MANUAL OF DIAGNOSIS AND THERAPY. Edited by C. E. Lyght and others. Rahway, New Jersey, Merck and Co., Inc., 1956. 1833 pages, 40 illustrations, 63 tables, thumb and general indexes. Price: \$6.75 (regular edition); \$9.00 (de luxe edition).

The ninth edition of this handy and popular manual of medical diagnosis and treatment, so useful to physicians in any field but particularly to the general practitioner, is the work of over 100 leading clinicians in the United States, Canada, and abroad. It has been ably edited by the editor and his staff so that a uniform presentation is skillfully accomplished.

Part I covers all sections of medicine, and it is a pleasure to record that ophthalmology is represented here, extraordinarily well and accurately. The definitions, classifications, signs and symptoms, etiology, pathology, diagnosis, laboratory findings, prognosis, and treatment of the various diseases and conditions to which the human body is subject are clearly exposed. More than 1,600 prescriptions are included, embodying the most up-to-date medical advances. Part II consists of chapters devoted to various procedures, routines, diets, and so forth. This is a most useful part for ready reference.

The section on the eye includes 40 pages. It is adequate and sound for a running reference knowledge of ophthalmology and seems to be up-to-date in a proper didactic fashion. It should interest and stimulate the general practitioner who is often not very interested in and a little afraid of ophthalmology.

The ophthalmologist will find the manual stimulating and most useful not only in his own field but also in all of the other sections, for after all he is an M.D., too.

The paper used is India and, although the text print is fine, it is not too difficult to read.
Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

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| 1. Anatomy, embryology, and comparative ophthalmology | 10. Crystalline lens |
| 2. General pathology, bacteriology, immunology | 11. Retina and vitreous |
| 3. Vegetative physiology, biochemistry, pharmacology, toxicology | 12. Optic nerve and chiasm |
| 4. Physiologic optics, refraction, color vision | 13. Neuro-ophthalmology |
| 5. Diagnosis and therapy | 14. Eyeball, orbit, sinuses |
| 6. Ocular motility | 15. Eyelids, lacrimal apparatus |
| 7. Conjunctiva, cornea, sclera | 16. Tumors |
| 8. Uvea, sympathetic disease, aqueous | 17. Injuries |
| 9. Glaucoma and ocular tension | 18. Systemic disease and parasites |
| | 19. Congenital deformities, heredity |
| | 20. Hygiene, sociology, education, and history |

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Blatt, N. and Athanasiau, M. **Anatomic relations between the optic canal and the sphenoid sinus.** *Ann. d'ocul.* 190:241-260, April, 1957.

The authors feel that many cases of retrobulbar neuritis are due to inflammation of the sphenoid sinus. To substantiate this hypothesis they made an anatomic study of 200 human skulls. Twelve representative photographs and line drawings illustrate the variations in the relationship between the bony optic canal and the sphenoid sinus. In many cases the wall of the canal is thin and shows dehiscences so that there is almost a direct communication between the canal and the sinus. This is particularly true when the sinus is larger than normal. In some cases one sinus may be in contact with both optic canals and therefore a unilateral sphenoid sinusitis may provoke a contralateral or bilateral optic neuritis. The authors conclude that certainly from anatomic evidence, the sphenoid sinus may be largely responsible for many cases of idiopathic retrobulbar neuritis. (12 figures, 12 references)

David Shoch.

Conrads, H. **Modification of the silver impregnation method according to Schultze-Gross.** *Arch. f. Ophth.* 158:506-508, 1957.

This is a purely technical note on the method mentioned in the title. (2 figures, 4 references) Ernst Schmerl.

Geniz Galvez, Jose M. **The morphologic basis of accommodation. The innervation of the ciliary muscle.** *Arch. Soc. oftal. hispano-am.* 16:1255-1287, Dec., 1956.

The objective of this study was to determine whether the sympathetic system participates in the innervation of the ciliary muscle and in the process of accommodation. The literature on the structure of the peripheral vegetative nervous system and on the innervation of the ciliary muscle is thoroughly reviewed. The ciliary muscle of various animals and man was examined by the procedures of Jabonero, Boeke, Wolter, and Castro as well as by an original technique. The data are reported and illustrated with photomicrographs. The present study shows that the innervation of the smooth muscle of the ciliary body is principally parasympathetic, but fibers which could be of a sympathetic nature were also seen. The

circular as well as the radial portions of the ciliary muscle have an identical innervation. This study does not confirm the opinion that one portion of the ciliary muscle is innervated by the parasympathetic through a branch of the third nerve and the other part is controlled by the sympathetic. This morphologic study confirms Meesman's opinion of functional unity of the ciliary muscle. (26 photomicrographs, 15 references)

Ray K. Daily.

Heydenreich, A. **The problem of the leukocytes in the cornea.** *Klin. Monatsbl. f. Augenh.* 130:512-522, 1957.

In order to clarify the hematogenous and the autochthonous theories of the origin of the leukocytes in the cornea, rabbits were made leukopenic with Trimitan. The corneas were then exposed to a variety of insults (ultraviolet light, incisions, chemical burns, heat, infections). In the treated animals the absence of leukocytes in the cornea was most conspicuous. This contrasted with the control animals which showed a marked leukocytic infiltration. These results speak for the hematogenous origin of the corneal leukocytes. (12 figures, 16 references)

Frederick C. Blodi.

Pau, H. and Conrads, H. **The cells of Langerhans and the nerves of the corneal epithelium.** *Arch. f. Ophth.* 158:427-433, 1957.

In 1868 Langerhans described cells of starlike shape occurring in the epidermis. He had been able to demonstrate them by using the method of gold impregnation. The cells were considered to belong to the nerve tissue of the body. The authors studied these cells in the corneas of men and rabbits using the Schultze-Gross silver impregnation method. They consider the cells of Langerhans to be a nervous epithelial plasmodium serving the epithelial

nerves in the same way as Schwann's cells do. (8 figures, 13 references)

Ernst Schmerl.

Unger, Hans-Hellmuth. **The structure of the filtering system of the chamber angle.** *Arch. f. Ophth.* 158:509-523, 1957.

From enucleated eyeballs which had been examined gonioscopically the author took segments of tissue from the region of the corneoscleral junction by means of meridional and equatorial incisions. With a 6-mm. trephine he also removed discs from the limbal region of all eyes with normal anterior segment which had been collected earlier and finally he studied histologically all available discs which had been removed by Elliot's trephine operation therapeutically.

The normal filtering system consists of several more or less separable lamellae which are more numerous at the spur than in the corneal periphery. Most of the lamellae end before they reach the cornea. The innermost lamella consists of a loose reticulum. The outer scleral lamellae, the greater part of the filtration system, have predominantly circular, but also sagittal lacunae. The inner wall of Schlemm's canal is a tissue woven of dispersed fibrils of larger fibers and has many large, round spaces. The preformed openings in the connective tissue are covered with endothelial cells. The insertion of the ciliary muscle in the lamellar system is characterized by arborization of the elastic end-fibers and a change of direction of 90°. (11 figures, 40 references)

F. H. Haessler.

Wolter, J. Reimer. **The endings of centrifugal nerve fibers in the blood vessels of the human retina.** *Arch. f. Ophth.* 158:524-530, 1957.

It can be shown histologically that the blood vessels of the human retina are supplied by centrifugal nerve fibers of the

nerve fiber layer and these fibers are of two distinguishable types. (9 figures, 11 references)

F. H. Haessler.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Ashton, N. **Experimental retrolental fibroplasia.** *Ann. Rev. Med.* 8:441-454, 1957.

The treatment of retrolental fibroplasia is now a closed chapter. However, the physiology of oxygen in the developing retina is quite different from that in any other tissue, and deserves further study. Such work can only be done on kittens, puppies, rats, or mice. Rabbits show no sign of retrolental fibroplasia.

Under direct observation, oxygen was found to constrict new capillaries so much that the retina turned white. Revascularization then came not from the disc, but from the persisting vascular trunks. From 12 to 24 hours of oxygen are required to do this. Oxygen less than 40 percent caused no harm, and CO₂ made no difference. Anoxia did not produce a retinopathy. If the retina is separated from its choroid for the experiment, the same retinal capillaries are not sensitive to excess oxygen. The perivascular capillary-free zone in the retina is narrowed in anoxia and widened in excess oxygen, indicating the extreme limits of a normal process. No other metabolic change in the retina has been found, such as oxygen consumption, anaerobic glycolysis, or succinic dehydrogenase. Paul W. Miles.

Boros, B. and Takats, I. **Comparative study of antihistaminics.** *Ophthalmologica* 132:386-395, Dec., 1956.

The antihistaminic effect of antistine was compared with that of a new agent called Synopen (manufactured by Geigy) on the isolated sphincter iridis of cattle or pigs. The typical effect of histamine

on this muscle is a marked contraction. The two antihistaminics differed in that antistine had the stronger immediate and Synopen the more lasting, contraction-inhibiting effect. The prolonged antiallergic effect of Synopen has been confirmed clinically in various allergic states. (6 figures, 12 references)

Peter C. Kronfeld.

Jaeger, W. and Seraphin, R. **Vascular reactions following intravenous injections of highly purified bacterial pyrogens.** *Arch. f. Ophth.* 158:449-461, 1957.

It had been found that intravenous injections of bacterial vaccine produce fibrinolysis. The question arose whether bacterial substances could be used to combat intraocular vascular occlusions. The authors injected highly purified pyrogens and studied the vascular reactions of the skin in a number of persons, using a special calorimeter. They found a pronounced vasodilatation, which however was preceded by a vasoconstriction for 30 to 60 minutes. The occurrence of the latter is considered undesirable. (4 figures, 3 tables, 29 references) Ernst Schmerl.

Nelken, E., Michaelson, I. C., Nelken, D. and Gurevitch, J. **Studies on antigens in the human cornea and their relationship to corneal grafting in man.** *J. Lab. & Cl. Med.* 49:745-752, May, 1957.

The cause of late clouding of successful corneal grafts is not known and since they are homografts the question has arisen whether the clouding results from an immunologic reaction between the tissues of donor and recipient. The existence of A and B antigen in human corneas corresponding to these antigens in the red blood corpuscles has been established by adsorption and elution experiments. It was not possible to show that the human cornea possesses Rh antigens. An immune reaction was found in patients who had

perforating keratoplasty with grafts from donors with incompatible blood groups. Antigens and antiB antibodies were found in human aqueous in individuals with high serum titer. F. H. Haessler.

Roberts, D. St. C. **Studies on the antigenic structure of the eye using the fluorescent antibody technique.** *Brit. J. Ophth.* **41**:338-347, June, 1957.

The antigen-antibody technique of Coons and Kaplan (1950) is a refined staining method. Fluorescein isocyanate is coupled to an antiserum and this is then used to treat fresh frozen sections of tissue. When an ultraviolet light is employed, the site of the antigen-antibody reaction is seen as a green discoloration. An antiserum was produced to rat glomeruli and this was found to react specifically with membranes in the ciliary body and iris. Small-vessel membranes in the retina, optic nerve and conjunctiva also reacted to this test. This specific staining would seem to suggest that a common antigen is shared by them and the glomerular basement membrane. (12 figures, 16 references) Lawrence L. Garner.

Toda, S. **Vitreous replacement as a treatment of experimental intraocular infection.** *Acta Soc. Ophth. Japan* **61**:568-581, May, 1957.

Rabbits were inoculated with staphylococcus into the vitreous. Then the vitreous was replaced with normal vitreous after a various period of time. A replacement within 19 hours after the infection showed a definite blocking effect on the inflammation. A combination of an injection of an antibiotic (5,000 units penicillin) into the vitreous with the vitreous replacement showed a tremendous effect, while simple antibiotic administration into the vitreous showed a poor effect. (10 figures, 7 tables, 30 references)

Yukihiko Mitsui.

Wolter, J. Reimer. **Changes of the neuroglia in advanced retinal degeneration.** *Klin. Monatsbl. f. Augenh.* **130**:498-511, 1957.

Twenty-six atrophic eyes were the basis of this study. Hypertrophy and proliferation of the astroglia represented the most conspicuous reaction. The neurons were destroyed and Müller's fibers remained passive. Pseudorosettes were found. The remaining nerve fibers were probably of the centrifugal type. (13 figures, 15 references) Frederick C. Blodi.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Alagna, G. and D'Aquino, S. **Ocular changes in selenium intoxication.** *Arch. di ottol.* **61**:55-71, Jan.-Feb., 1957.

Ten rabbits were given $\frac{1}{2}$ mg. of sodium selenite daily for 4 days, 1 mg. for 4 days more, and $1\frac{1}{2}$ mg. for an additional 4 days. Ten other rabbits were given 1 mg. sodium selenite daily intravenously. Changes were observed in the liver (chronic cirrhotic hepatitis, necrotic-hemorrhagic hepatitis), the spleen and the eyes (inflammation and degeneration of iris and ciliary body, cataract of the endocrine type, diffuse degeneration of choroid and retina, and primary degeneration of the optic nerve fibers). (6 figures, 30 references) John J. Stern.

Ambrosio, A. **The distribution of the carbonanhydrase activity in the lens.** *Arch. di ottol.* **61**:41-54, Jan.-Feb., 1957.

Carbonanhydrase is shown to be present in the epithelium, the capsule, the cortex and the nucleus. (2 tables, 87 references) John J. Stern.

Barraquer, J. and Canadell, J. M. **The exophthalmogenic factor in blood serum.** *Ophthalmologica* **132**:374-381, Dec., 1956.

In 1945 Albert reported the production

of exophthalmus in the salt water fish *Fundulus heteroclitus* by repeated injections of anterior pituitary lobe extracts. In 1953 Dobyns and Wilson produced the same phenomenon of exophthalmus in the same fish by injection of serum from patients with hyperthyroidism and exophthalmus. The authors of the paper under review have used this phenomenon as a test in the study of various types of exophthalmus. *Fundulus* was not available in Spain, but another species, *Carassius auratus*, proved just as suitable and easy to keep in four-gallon tanks in groups of four fish, three to receive the injection and one to serve as control. The injections were made into the coelom, in doses of .25 cc., and repeated every six hours until a total dose of 1.5 or 2 cc. was reached. Fresh serum without any preservative or other chemical treatment was used. If the serum contained the exophthalmogenic factor, the eyes of the fish started to protrude after the fourth injection. In a small series of patients presenting various types of exophthalmus there seemed to be a fair degree of correlation between a positive "Carrassius test" and clinical findings of thyroid-pituitary disease. (8 figures, 5 references)

Peter C. Kronfeld.

Boros, B. and Takatz, I. **Determination of acetylcholine in corneal transplants.** Arch. f. Ophth. 158:416-428, 1957.

The authors determined the amount of acetylcholine in 30 autotransplants of rabbits, using frogs' hearts. They compare good and clear transplants with those which had become cloudy and found the amount of acetylcholine the higher the better the conditions of the transplants. The reinnervation, however, did not seem to be disturbed in either kind of transplants. The amount of acetylcholine varied with the time of determination after transplantation and was found

within a range of from nothing to 8.3 γ per gram of tissue. (6 figures, 1 table, 19 references)

Ernst Schmerl.

Brunish, R. **The protein components of human tears.** A.M.A. Arch. Ophth. 57: 554-556, April, 1957.

Electrophoretic tests on tears disclosed the presence of albumin, lysozyme, and three globulins. Irritant-induced tears contain less albumin than emotionally induced tears. (3 figures, 1 table, 4 references)

George S. Tyner.

Cherednichenko, V. **The action of novocaine block with some other influences on the nervous system in inflammatory processes of the eye.** Vestnik oftal. 3:3-5, May-June, 1957.

A study was made of the influence of factors depressing and stimulating the central nervous system. Five series of experiments on rabbits were made. The inflammation of the anterior segment of the eye was produced by introduction of 0.6 mg. of trypsin into the anterior chamber. In the first series (40 experiments), the action of retrobulbar blockade with a solution of 1 percent novocaine on the course of the inflammatory reaction in the iris and ciliary body was studied. The experiments and the pathologic picture of the enucleated eyes showed that the retrobulbar injection of 1 cc. of 1 percent solution of novocaine halted the development of the inflammation; the exudate in the anterior chamber and the edema of the iris and ciliary body decreased. This was more pronounced in a mild inflammatory process.

In the next series of 80 experiments trypsin was introduced into the anterior chamber together with novocaine (1 percent solution). The inflammatory reaction was also decreased in this experiment. The depressing action of novocaine on the inflammation is possibly connected

with its action on the neuro-receptory apparatus of the iris and ciliary body. In the fourth series (54 experiments) the influence of therapeutic sleep was studied by subcutaneous injections of urethane and veronal. A decrease of the inflammatory reaction was observed and ascribed to a slowing of the activity of the central nervous system. There was a decrease of the edema and hyperemia of the iris blood vessels. The decrease of the permeability of the vessels decreases the exudation of fibrinogen; photophobia and epiphora disappeared.

In the last series of 54 experiments, the central nervous system was stimulated by subcutaneous injections of 0.02 cc. of caffeine. The inflammatory reaction was increased by this procedure.

The author concluded that: 1. the inflammatory reaction in the eye develops by reflex participation of the nervous system (including the cerebral cortex), 2. the retrobulbar injection of novocaine checks the development of an aseptic reaction in the eye, 3. the therapeutic sleep decreased the inflammation in the chamber of the eye, and 4. the stimulation of the cortex with caffeine increases the inflammatory reactions. Olga Sitchevska.

Cibis, P. A., Constant, M., Pribyl, A. and Becker, B. **Ocular lesions produced by iodoacetate.** A.M.A. Arch. Ophth. 57:508-519, April, 1957.

This study was undertaken on rabbits to compare the ocular lesions produced by iodoacetate with those produced by radiation. Both inhibit the activity of certain enzyme systems. The histologic changes induced by iodoacetate involved the corneal endothelium, ciliary epithelium, retina and lens in a manner similar to the sequellae of radiation. (14 figures, 4 tables, 21 references) George S. Tyner.

Ciusa, W., Cristini, G. and Garutti, M. A. **Nicotinic acid metabolism in the**

lens, the aqueous and the vitreous. Klin. Monatsbl. f. Augenh. 130:488-491, 1957.

Nicotinic acid occurs in the aqueous. In the lens the amine is formed and the latter is finally transformed into the methyl derivative. Similar changes occur in the vitreous. A transformation into pyridon does not occur in the eye. The analyses were done on bovine eyes. (1 table, 12 references) Frederick C. Blodi.

Cuccagna, F. **The effect of Elienene on the adaptation in myopia and retinitis pigmentosa.** Arch. di ottal. 60:311-321, Nov.-Dec., 1956.

Elienene is the dipalmitate ester of xanthophyll, marketed under the name of Adaptinol. It was administered to six myopes and four patients with retinitis pigmentosa for two weeks (10-20 mg. per day). Examination with the Goldman-Weeckers adaptometer demonstrated an improvement of the light sense, most pronounced in cases with only slight anatomic changes in the retina. (2 figures, 14 references) John J. Stern.

Dardenne, U., Leydhecker, W. and Helferich, E. **The cholinesterase of the iris in man and cattle.** Arch. f. Ophth. 158:434-438, 1957.

The authors studied the problem whether the iris of man and cattle contains specific as well as unspecific cholinesterase. They describe their experimental procedures in detail and give the following summary. The iris of men and cattle contains specific cholinesterase only. There were no indications of the presence of pseudo-cholinesterase. The iris of cattle can be considered a satisfactory substitute for human iris with respect to enzymatic studies. (3 figures, 17 references) Ernst Schmerl.

Fujiu, K. **Histochemical studies of cholin in the retina.** Acta Soc. Ophth.

Japan 61:179-190 and 406-417, Feb. and April, 1957.

This is a histochemical study of the cholin substances in the animal retina. Fujiu first studied the frog retina and demonstrated that the cholin substances in the outer segment of the visual cells increased and decreased respectively in light and dark adaptation. Then he used chicken and guinea pig as the representatives of the cone and rod retina. It was then demonstrated that the cone retina contained less cholin than the other and that the decrease and increase in the cholin by dark and light adaptation respectively occurred rapidly (in two or three minutes) in the cone retina, while it occurred slowly (in 10 to 15 minutes) in the rod retina. He considered the fact to indicate that there was a more rapid photosynthesis and decomposition in the cone retina than in the other.

Next he studied in frogs the effect of methanole and quinine; a complete inability to increase cholin by light adaptation resulted. Fujiu suggests that acute toxic amaurosis might be due to a disturbance in the retinal phosphatide by the toxines. He further showed that such inhibitors of rhodopsin regeneration as cocaine, potassium cyanide and monoiodic acetate had the same effect as amaurotic agents for the cholin in the retina. Finally, he studied the effect of carbon tetrachloride on the cholin. A single administration of this agent showed no effect. Repeated, prolonged administrations, however, caused a decrease in the cholin independently of the status of the adaptation. He suggests that this might not be due to a direct action of the agent on the retina but be secondary to a disturbance in liver function. (14 figures, 10 tables, 86 references) Yukihiro Mitsui.

Hager, G. **Adjustment of gaze in physiologic ageing.** Arch. f. Ophth. 158:598-604, 1957.

The apparatus is described with which it was possible to demonstrate a statistical reduction in modification of gaze with physiologic ageing in 300 subjects with normal eyes. (2 figures, 2 tables, 16 references) F. H. Haessler.

Hagihara, T. **Pupillary movement of removed eye. Part IV.** Acta Soc. Ophth. Japan 61:391-401, April, 1947.

This is an analysis on the mechanism of pupillary reaction in the removed eye. When the aqueous of the removed eye made miotic by light or high temperature, was injected subconjunctivally into another animal, it caused a miosis. Then such aqueous or an iris emulsion of the same eye was added to the medium of Magnus' measurement and the movement of the intestine was increased. The increase was impeded by an addition of atropine. Hagihara suggests that the substance which brings about the pupillary reaction in the removed eye is acetylcholin or a like substance. (23 figures, 2 tables, 18 references) Yukihiro Mitsui.

Hamada, K. **A study of the blood pressure in the ciliary vein.** Acta Soc. Ophth. Japan 61:109-112, 347-355 and 455-460, Feb., April and May, 1957.

To measure the blood pressure in the ciliary vein, Hamada designed a new electric compression apparatus, which had the same principle as a millimeter and in which the hand of the meter acted as a compression lever. He first measured the ciliary vein pressure in 186 normal eyes and gave the figure between 5.8 and 17.9 mm. Hg with an average of 10.4 mm. Hg. The frequency of distribution of the pressure was not binomial but the pressure showed a correlation with the ocular tension. A similar measurement was then performed in 52 glaucomatous eyes. Then the venous pressure was in the range of 7.7 and 21.8 mm. Hg, with an average of 14.0 mm. Hg. There was no correlation

between the venous and ocular tension in glaucomas but both tensions showed an analogous diurnal variation. In contrast to Duke-Elder, Hamada found that the ocular pressure was consistently higher than the venous pressure, not only in the ascending phase but also in the descending phase.

In glaucomatous eyes the venous pressure showed a tentative rise above the original level after the termination of an eyeball compression by 50 gms. for 10 minutes, while it did not occur in normal controls. He also measured the venous pressure in some other ocular conditions and found that the venous pressure was apt to be low in Eales' disease, senile cataract, optic nerve atrophy and pigmentary degeneration of the retina, and high in angiosclerosis and central serous retinopathy. (8 figures, 24 tables, 83 references) Yukihiko Mitsui.

Hiyama, H. and Ikui, H. **Experimental and clinical studies on intraocular migration of tetracycline by systemic administration.** *Acta Soc. Ophth. Japan* 61:520-531, May, 1957.

In rabbits tetracycline given by mouth does not appear in the aqueous at a concentration higher than 1 γ /ml. This concentration is obtained by giving 250 mgs. at one time and an increase in the dosage over this level does not increase the concentration in the blood and aqueous. However, when the agent is given intravenously in a dosage of 100 mgs., a concentration as high as 18 γ /ml. can be obtained in the aqueous. In both cases the aqueous concentration is about one tenth of the blood concentration. Similarly, 10 to 20 times greater concentration of the antibiotic can be obtained in the cornea, vitreous and uvea by an intravenous administration than by an oral administration. In a case of endophthalmitis in man, an intravenous injection of this antibiotic

showed an excellent effect. (2 figures, 3 tables, 14 references) Yukihiko Mitsui.

Ikuta, M. **A study on the relation of the aqueous pressure to the vitreous pressure.** *Acta Soc. Ophth. Japan* 61:589-613, May, 1957.

This is a study of the relation between the aqueous and vitreous pressure in rabbits. The pressures are measured by a manometer. Ikuta states that the change in the aqueous pressure induced by some autonomic agents is not always parallel to that in the vitreous pressure. After an injection of the air into the vitreous or a vitreous puncture, a greater increase or decrease of pressure, respectively, results in the vitreous than in the aqueous. An electric stimulation of the sympathetic center in the midbrain causes a greater increase of pressure in the aqueous than in the vitreous. Ikuta concludes that the non-parallelism may be explained by a buffer action of the lens-iris diaphragm. He also states that Bellow's observation of a high vitreous pressure by a standing aqueous drainage was not observed by him. (14 figures, 14 tables, 54 references) Yukihiko Mitsui.

Leydecker, W., Dardenne, U. and Helferich, E. **Combined use of miotics.** *Arch. f. Ophth.* 158:439-448, 1957.

Prostigmin is said to inhibit cholinesterase reversibly while mintacol (diethyl-p-nitrophenyl-phosphate) destroys it irreversibly. In the opinion of some authors reversible inhibition interferes with irreversible inhibition. The authors of the present study examined this question experimentally using Ammon's method and the Warburg apparatus. They found that prostigmin protects the enzyme against the action of mintacol when the inhibitor together with the iris tissue is incubated first and acetylcholine is added thereafter. Both inhibitors show a combined action

when they and acetylcholine are simultaneously added to the iris tissue. The authors reach the conclusion that the clinical simultaneous use of prostigmin and mintacol may produce a stronger decrease of the ocular tension than when given separately. (5 figures, 2 tables, 13 references)

Ernst Schmerl.

Lucas, D. R., Newhouse, J. P. and Davey, J. B. **Experimental degeneration of the retina. The lesion produced by bromoacetate.** *Brit. J. Ophthalm.* 41:313-316, May, 1957.

During a study of the effect of various thiol reagents upon the retina, it was observed that bromoacetate, when injected intravenously, caused some histologic and ophthalmologic changes. With the ophthalmoscope a retinal haze was seen immediately, followed after about ten days by a pigment clumping in the mid-ventral area. There were no histologic changes after a single small dose of the drug, but greater doses resulted in localized loss of visual cells in the area centralis with much pyknosis of cell nuclei. These changes seem to be a direct effect of the drug. (5 figures, 8 references)

Morris Kaplan.

Minoda, Y. **Effect of antibiotics on some fungi isolated from ocular lesions.** *Acta Soc. Ophthalm. Japan* 61:562-567, May, 1957.

Candida albicans, *Aspergillus fumigatus*, *Penicillium* sp., and *Acremonium* sp. isolated from ocular lesions, particularly from corneal infections, were tested for the sensitivity to some antibiotics. Trichomycin, an antibiotic established in Japan, was definitely effective for *Candida* but not for other fungi. The minimum inhibitory concentration of trichomycin to *Candida* was roughly 6 mcg/ml. (4 figures, 1 table, 23 references)

Yukihiko Mitsui.

Mishima, S. **The sympathetic innervation of the rabbit cornea.** *Acta Soc. Ophthalm. Japan* 61:137-143, Feb., 1957.

The effect of sympathetic denervation and stimulation for the corneal epithelium was studied in rabbits, using the mitotic rate of the epithelial cell as the indicator. After a removal of the superior cervical ganglion, a slight increase in the mitosis resulted in 6 to 12 hours, which was followed by a lasting decrease during the next several days. When the ganglion was electrically stimulated, a definite decrease in the mitosis of the corneal epithelium followed. (4 figures, 3 tables, 12 references)

Yukihiko Mitsui.

Münch, W. **Experimental studies of the behavior of acid-soluble compounds of phosphorus, particularly adenosintriphosphate (ATP) and adenosindiphosphate (ADP) in the corneal tissues.** *Arch. f. Ophthalm.* 158:532-557, 1957.

ATP and ADP are of fundamental importance in the transport of energy in the living cell. Most of the energy set free in cell respiration is immediately absorbed in synthesis of energy-rich phosphates which store the energy. The breaking down of these energy-rich phosphates is a direct source of energy for the activities of the cell. The author's investigation concerns details of this process. Fresh eyes of calves and rabbits were used. (26 tables, 111 references)

F. H. Haessler.

Ogata, S. **Antibiotic allergy with special reference to the cutaneous and instillation reaction.** *Acta Soc. Ophthalm. Japan* 61:538-548, May, 1957.

A cutaneous reaction is positive only in 30 percent of the subjects allergic to penicillin. A single instillation of the agent into the eye causes an ocular reaction only in 17 percent though a repetition of the instillation increases the positivity. However, in subjects anaphylactic

to penicillin, an instillation reaction is almost always positive. The reaction is prompt and definite and, in addition, harmless. A cutaneous reaction in such cases often causes severe systemic reaction. Ogata recommends a trial of instillation reaction before penicillin injection for prevention of anaphylactic shock. A solution in a concentration of 100 U/ml is adequate.

Leucomycin, an antibiotic established in Japan, having a spectrum similar to that of erythromycin, has a strong antigenicity. It very often causes blepharoconjunctivitis after prolonged instillation. A cutaneous reaction was positive in all of the allergic cases. Systemic sensitization occurs easily with this antibiotic even by a topical instillation into the eye. The provocation tests with these antibiotics in the eye or skin cannot be impeded by a simultaneous application of cortisone or antihistaminic agents. (5 figures, 4 tables, 12 references)

Yukihiko Mitsui.

Orlowski, W. J., Wekka Z. and Nowakowski, W. **Chemical composition of aqueous in experiments with X-ray irradiation. I. Level of sodium and potassium.** *Klinika Oczna.* 27:15-25, 1957.

The authors state that the behavior of sodium and potassium ions in the aqueous is not well known. Experiments were made on 29 rabbits measuring concentration of potassium and sodium ions in the aqueous and in the serum. Average sodium concentration in the serum is 320.7 percent mg. and in the aqueous 384.4 percent mg. Average potassium concentration in the serum was 22.1 percent mg. and in the aqueous 17.9 percent mg. Additional 12 rabbits were subjected to X-ray irradiation of 1500 r. to the right eye and of 3000 r. to the left eye. Repeated determination of sodium and potassium levels showed that within two and one-

half hours after irradiation the levels of both ions were higher than before and within two weeks were gradually returned to the usual concentration. It was noticed that after four and six weeks after irradiation there were again changes in the aqueous concentration of sodium and potassium. (2 figures, 4 tables, 18 references)

Sylvan Brandon.

Pedler, Christopher. **Studies on developing retinal vessels. IV. Effect of ionizing radiation.** *Brit. J. Ophth.* 41:179-181, March, 1957.

It has been established that hyperoxia of the vessels of the immature retina will result in permanent obliteration of these vessels within 8 to 36 hours. It has been suggested that ionizing irradiation might have similar or related effects and that both these actions may be brought about by a breakdown in the enzyme system of the retina. In this series of experiments kittens' eyes were exposed to cobalt 60 radiation and both the immediate and the delayed effects were studied. No abnormal reaction was found in the vessels. It is concluded that the animal's biochemical system known to be affected by irradiation is not involved in the vaso-obliterative reaction. (1 table, 14 references)

Morris Kaplan.

Perkins, E. S. **Influence of the fifth cranial nerve on the intraocular pressure of the rabbit eye.** *Brit. J. Ophth.* 41:257-300, May, 1957.

Rabbits were used to determine the effect of stimulation of the fifth cranial nerve on the ocular tension. Mechanical stimulation of the intact nerve on the same side brought about a contraction of the pupil, a rise in temperature of the ciliary region, a rise in intraocular pressure and an increase of protein in the aqueous. In the contralateral eye there was no contraction of the pupil, but a rise

in ocular tension occurred in 15 percent of the animals. Section of the nerve produced the same immediate effects as stimulation. Stimulation of the nerve caused a vasodilatation of the episcleral vessels of the ipsilateral eye but no change in the choroidal vessels. When the nerve had been sectioned two to four weeks previously, stimulation of the cranial portion of the nerve caused no changes in the pupil or the tension. Histologic preparations of the nerves previously sectioned showed degenerated fibers in that part of the nerve central to the section. These results tend to show that the fifth cranial nerve in rabbits can mediate impulses for raising tension but this does not necessarily show that the nerve takes an active part in the regulation of tension. (17 figures, 9 tables, 95 references)

Morris Kaplan.

Roper-Hall, M. J. **Oxyphenonium as an atropine substitute in ophthalmology.** *Brit. J. Ophth.* 41:238-242, April, 1957.

Oxyphenonium was used in a series of 57 patients as a substitute for atropine because of sensitization to the latter. It is particularly useful in those patients that have shown irritation to atropine or to atropine substitutes. No evidence of irritation or sensitization was noted in this series; some stinging was complained of by some and can be averted by buffering. The onset of cycloplegia is somewhat faster than that of atropine and the duration of its effects is considerably longer. (1 figure, 7 references)

Lawrence L. Garner.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Best, W. and Bohnen, K. **The "off-effect" in the electroretinogram in man.** *Arch. f. Ophth.* 158:577, 1957.

In the electroretinogram the off-effect

is demonstrable only during light adaptation and not during dark adaptation. The off-effect engrafts itself upon the on-effect and gives rise to a combined positive wave. The off-effect becomes greater with increase of duration of the stimulus and in certain conditions of procedure it consists of several positive waves. Most of these recordings were made on three subjects, a few on twenty. The essential nature of the off-effect is discussed. (5 figures, 2 tables, 14 references)

F. H. Haessler.

Bonnet, R. **Optical theory of and adaptation to contact lenses.** *Ann. d'ocul.* 190:281-292, April, 1957.

After a brief historical review the author gives the optical indications for contact lenses. These are high degrees of ametropia, particularly irregular astigmatism, keratoconus and aphakia, and anisometropia. He analyses the effect of contact lenses on the optics of the eye in an abbreviated mathematical fashion and points out the necessary physical characteristics to insure wearability. Primarily the author makes use of the fluorescein pattern to fit the lens properly. (12 figures)

David Shoch.

Jayle, G.-E., Ourgaud, A.-G., Lozivit, P. and Aubert, L. **Is there a second inflection point in the dark-adaptation curves?** *Ann. d'ocul.* 190:213-219, March, 1957.

The authors studied dark adaptation in 36 normal subjects. In several they were able to show a second point of inflection in the rod portion of the curve. It occurs after about 14 minutes of dark adaptation under the conditions maintained by the experimenters. (4 figures, 5 references)

David Shoch.

Lewinski, Horst. **The objective determination of visual acuity.** *Arch. f. Ophth.* 158:578-597, 1957.

The visual acuity of 246 abnormal eyes was measured with the instrument of Nicolai which is based on Günther's principle. By making it possible to vary the distance between eye and object the range was increased so that acuity as low as 0.5 could be measured. It emerged that the objective visual acuity can be stated with a 75 percent probability. A new simple principle for providing the stimulus was designed in which a variable slit rather than a checker board design is related to the resolving power of the eye. (4 figures, 11 tables, 15 references) F. H. Haessler.

5

DIAGNOSIS AND THERAPY

Dalma, J. and Victoria, V. **Report on the results obtained with pyretotherapy in 51 cases of Chagas' disease.** Arch. oftal. Buenos Aires 32:55-58, Feb., 1957.

The unresponsiveness of trypanosomiasis americana—a condition in whose early stages a pronounced, hard, congestive edema of both lids of one eye occurs, together with swelling of the associated lymph nodes—to practically all forms of treatment is well known. Since blood agar cultures of the protozoon are sterilized in 85 hours when kept at 39°C. and in 18 hours when maintained at 40°C., it was thought that by raising the patient's temperature with the aid of fever-producing agents (especially with intravenous injections of typhoid vaccine), an amelioration could be achieved. The results were dramatic: considerable improvement took place in a matter of hours in all the 52 patients so treated and was shortly followed by a clinical cure; in 22 of the 24 which could be observed for no less than one year, the xenodiagnostic test became consistently negative, while in 15 of 33 control cases submitted to other forms of therapy it remained positive at the end of that period. (9 references)

A. Urrets-Zavalía, Jr.

Filgueira, E. L. and Castillo, J. L. **Photography of the eye through the corneal microscope.** Arch. oftal. Buenos Aires 32:62-65, Feb., 1957.

Both a 35 mm. camera and a $2\frac{1}{4} \times 3\frac{1}{2}$ inch bellows camera were adapted to one ocular of the Goldmann microscope; observation and focussing were made through the other one. With film speed ratings of about 100 A.S.A., exposure times of $\frac{1}{8}$ to $\frac{1}{2}$ seconds with the miniature and of $\frac{1}{2}$ to 2 seconds with the larger camera were needed. The results did not seem to be exceedingly good. (7 figures)

A. Urrets-Zavalía, Jr.

Jayle, G.-E., Aubert, L., Boyer, R. and Camo, R. **A comparative study of photopic perimetry, mesopic and scotopic campimetry in a case of cataract.** Ann. d'ocul. 190:206-212, March, 1957.

The authors continue their studies on visual fields with intermediate illumination (mesopic campimetry). In a patient with a unilateral cataract they found that this type of field testing gave the most accurate results. Dark adaptation curves are also given. (3 figures) David Shoch.

Jayle, G.-E., Blet, G., Aubert, L., Boyer, R., and Lozivit, P. **Preliminary note on mesopic campimetry in ocular pathology.** Ann. d'ocul. 190:108-116, Feb., 1957.

The authors feel that visual field studies done with bright illumination are not delicate enough to pick up fine changes. Also those done under scotopic conditions are difficult for the patient because of the physiologic central scotoma produced. They therefore propose an intermediate illumination (mesopic campimetry) and demonstrate its value in a case of macular choroiditis. (3 figures)

David Shoch.

Julinskaya, A. **Streptomycin and Ftivazid in the treatment of tuberculosis of the eye.** Oftal. Zhurnal. 1:10-14, 1957.

Streptomycin and Ftivazid (a derivative of isoniazide) were used on 120 patients with various forms of tuberculosis of the eye in the Lvov Eye Clinic during the years 1952 to 1954. Streptomycin was given intramuscularly, 20 to 40 million units, during the course of treatment; also by retrobulbar or subconjunctival injections daily or every other day in doses of 25 to 50 thousand units, the number of injections being from 15 to 30. Drops and ointment of streptomycin were used in affections of the anterior segment of the eye.

Of 73 patients who were given streptomycin 32 had keratitis, 9 had kerato-scleritis, 15 uveitis, 13 chorioretinitis, and 4 retinal detachment. Of the treated patients 69 improved and no recurrence was observed.

Ftivazid was applied since 1953; 17 patients were given only Ftivazid tablets and 30 combined therapy of streptomycin and Ftivazid. Only one patient showed intolerance to Ftivazid. Best results were obtained with the combined treatment of streptomycin and Ftivazid in the treatment of tuberculous diseases of the eye. Ftivazid, because of the simplicity of its use and absence of side effects, is a valuable remedy, particularly in children and undernourished patients.

Olga Sitchevska.

Merz, Marian. **Isotopes in ophthalmology.** *Klinika Oczna* 27:63-72, 1957.

The author gives basic information about isotopes in general and a little more about those which are used in medical research or treatment. The use of P^{32} is described in detail for diagnosis of intra-ocular tumors. The therapeutic value of isotopes is described and compared. The method of application is described briefly and a few conditions are listed in which treatment with isotopes is effective. (4 figures, 40 references.)

Sylvan Brandon.

Pünder, Hermann. **Treatment of lime burns.** *Klin. Monatsbl. f. Augenh.* 130: 536-539, 1957.

The experiences are based on 88 cases. The conservative treatment is preferred. In only ten patients was a mucosal graft performed and the result was not superior to the medical treatment. The author advises the subconjunctival injection of Priscoline, patching, heat and later vitamine A ointments. (22 references)

Frederick C. Blodi.

Rizzo, Paolo. **The therapy of asthenopia with a collyrium of digitalis.** *Arch. di ottal.* 61:19-40, Jan.-Feb., 1957.

A collyrium containing 0.2 percent digitalis was used for two weeks by 306 patients with asthenopic complaints. Favorable effects were observed in incipient presbyopia, refractive errors and general asthenopia. A partial effect was seen in patients with asthenopia and muscular disturbances. The results were negative when the disturbance was due to a disturbance in a distant focus (sinuses, ovaries). Patients complaining of disturbances when viewing television also benefited. The subjective improvement was affirmed in over half of the patients by checking the punctum proximum. The effect is explained by the cholinergic effect of digitalis and improvement of the blood supply to the ciliary body. (3 tables, 55 references)

John J. Stern.

Rutkowski, Slawomir. **Problems of corneal transplantation. I. Mechanism of corneal trephination and some new trephines.** *Klinika Oczna* 27:1-8, 1957.

Proper excision of the corneal transplant may make the difference between successful operation or failure. Premature loss of anterior chamber and injury to the iris and the lens may complicate the operation. The author describes the actions of various types of trephines. He experimented on animal eyes and came to

the conclusion that there is no trephine which insures the safety of the iris or the lens. Trephines with micrometric arrangement are the best because they make it possible to operate on eyes with shallow anterior chamber, opaque cornea and when lamellar transplantation is used. Complete excision of the cornea with a trephine should not be attempted because of possible complications; the excision can be completed with scissors. The best results are obtained when trephines are sharpened for each operation. (4 figures) Sylvan Brandon.

de Saint-Martin, R. **Maximal preoperative hypotony in surgery of the eyeball.** *Ann. d'ocul.* 190:165-186, March, 1957.

In 114 surgical operations on the eye (96 cataract extractions and 18 antiglaucoma procedures) the author added hyaluronidase to the retrobulbar anesthetic mixture. He finds that this softens the eye markedly and makes the operation safer. Operative and postoperative complications were decreased, and there were no serious sequelae that could be traced to the use of hyaluronidase. (3 tables) David Shoch.

Samuelly, C. **The topical use of prednisolone in ophthalmology.** *Arch. di ottal.* 61:81-94, Jan.-Feb., 1957.

The results of prednisolone therapy in 26 cases of conjunctivitis, scleritis, iritis and keratitis are reported. This product was found to be superior to cortisone. (16 references) John J. Stern.

Shubova, T. and Konchakova, M. **The value of eye signs in the diagnosis of multiple sclerosis.** *Vestnik oftal.* 3:24-25, May-June, 1957.

Sixty-six patients, aged 20 to 55 years, who had multiple sclerosis were studied. The authors state that among the eye signs diplopia, nystagmus (in 48 pa-

tients), spasm of the accommodation, and anisocoria (in 17 patients) were helpful in making a correct diagnosis of multiple sclerosis. In 26 patients there was atrophy of the optic disc which usually was accompanied by faulty color fields. In many patients there were narrow arteries and periphlebitis of the retinal veins. Sudden loss of vision occurred in eight patients. The vision remained from 0.7 to 1.0 in 53 patients. In two patients there was hemianopsia, in one a ring scotoma, but as a rule the visual fields remained normal despite low visual acuity, which might be an aid in the differential diagnosis of the disease.

Olga Sitchevska.

7

CONJUNCTIVA, CORNEA, SCLERA

Aberastain, Tomas G. **Marginal dystrophy of the cornea (Terrien's disease): report of two cases.** *Arch. oftal Buenos Aires* 32:9-12, Jan., 1957.

Two instances of this rare condition are described (one in a 43-year-old man and one in a 50-year-old woman), where the cornea exhibited a large marginal ectasia secondary to localized thinning of the anterior stromal layers. Vision was reduced in proportion to the gravity of the corneal deformity. (6 figures, 1 table)

A. Urrets-Zavalía, Jr.

Aichmair, Hermann. **Necrotizing conjunctivitis and keratitis in agranulocytosis.** *Klin. Monatsbl. f. Augenh.* 130:529-533, 1957.

The inflammation occurred in the left eye of a 58-year-old woman who died one week later. She had had rheumatoid arthritis and took Butazolidin and similar drugs. (1 figure, 8 references)

Frederick C. Blodi.

Böke, Wilhelm. **Critical remarks concerning the term "keratoconjunctivitis ec-**

zematosa." *Klin. Monatsbl. f. Augenh.* 130:533-536, 1957.

This term should be confined to the keratitis (often superficial and punctate) and conjunctivitis which accompanies various forms of true eczema and four such patients are briefly discussed. The term should not be used interchangeably with "phlyctenular keratoconjunctivitis." (10 references) Frederick C. Blodi.

Escapini, H. **Pathology of the human corneal graft.** *Arch. oftal. Buenos Aires* 32:31-48, Feb., 1957.

This paper is a slightly enlarged, though essentially unmodified, version of the report presented by the author to the XVII International Congress of Ophthalmology. Sixteen eyes, which had previously undergone at least one partial penetrating corneal transplant, were studied; the graft was opaque in 15 and clear in one. The view is held that no substitution of tissues takes place either in transparent or in cloudy grafts; only in cases with gross opacification and vascularization does an invasion by elements from the host occur. This conclusion is in keeping with that reached by Babel in a similar study (cf. Babel, J. *Les processus anatomiques de cicatrisation des greffes de la cornée.* Basel, S. Karger, 1950). (11 figures, 10 references)

A. Urrets-Zavalía, Jr.

Etienne, R. and Moreau, P.-G. **Nodular corneal dystrophy of Salzmann.** *Ann. d'ocul.* 190:187-205, March, 1957.

The authors review the initial reports of Salzmann's dystrophy and add seven cases to the literature. They make several important points. First the disease is a postinflammatory one and most often follows a phlyctenular keratoconjunctivitis. It is more common in women but is definitely not hereditary. Secondly, the pathologic process is limited to the

epithelium and in each of the four specimens examined Bowman's membrane was intact throughout. This is in contrast to previous studies in which an absence of Bowman's membrane was reported. Thirdly, the ideal treatment for eyes with visual loss is a lamellar corneal transplant. Patients without visual loss or discomfort should have no surgical treatment. (5 figures, 15 references)

David Shoch.

Riedl, Sabina. **Aneurysms of conjunctival vessels in diabetes.** *Klinika Oczna* 27:43-46, 1957.

Changes in the blood vessels of the conjunctiva were investigated in 60 diabetics and were compared with findings in 90 normal subjects. Aneurysms were found in 47 percent of diabetics and in 18.8 percent of nondiabetics. In nondiabetics aneurysms were found more frequently in hypertensive subjects. No relation was found between the presence of retinopathy and the appearance of conjunctival aneurysms. Sex did not influence their frequency. The presence of aneurysms is not pathognomonic of diabetes but a suggestive sign. (2 references)

Sylvan Brandon.

Rzehulka, Gertraud. **Zinc chloride burn of the eye.** *Klin. Monatsbl. f. Augenh.* 130:539-542, 1957.

A 69-year-old woman dropped a 50-percent zinc chloride solution into her left eye. The solution was erroneously prepared instead of a 1-percent solution. A severe burn of the cornea and the bulbar conjunctiva followed. The eye was treated with ammonium tartrate and cortisone. Final vision with correction was 6/8. (1 figure, 8 references) Frederick C. Blodi.

Segal, P., Freytag, T. and Czechowska, Z. **Use of plastic material in experimental wounds of rabbits' sclera.** *Klinika Oczna* 27:9-13, 1957.

Experiments were conducted to determine the advisability of using plastic implants to cover the loss of tissue. Pieces of sclera were removed from rabbits eyes varying from 5 by 5 mm. to 10 by 13 mm. Thin acrylic plate was sewed in with catgut. In half of the eyes the plastic was extruded in two to three months. Microscopic examination showed mild foreign body reaction, less intense than around the sutures. The healing of the sclera proceeded unhindered under the plastic cover. The author feels that in cases where plastic plates were extruded the tissue covering them was relatively thin. (6 figures, 4 references) Sylvan Brandon.

Tsutsui, J., Furusawa, T., Tsuji, S. and Takeda, S. **Development of immunity by repeated infection of trachoma.** A.M.A. Arch. Ophth. 57:577-584, April, 1957.

This study bears out the contention that injection with the trachoma virus does not confer an immunity sufficient to prevent reinfection after a clinical cure. (1 figure, 1 table, 13 references)

George S. Tyner.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Bronstein, M. **Vogt-Koyanagi-Harada disease.** A.M.A. Arch. Ophth. 57:503-507, April, 1957.

A 36-year-old white woman with combined manifestations of Vogt-Koyanagi and Haradas disease is reported. Long term prednisolone therapy (approximately two years) resulted in a final outcome of 20/30 vision in both eyes. (4 figures, 10 references)

George S. Tyner.

9

GLAUCOMA AND OCULAR TENSION

Birge, Henry L. **Prodromal malignant glaucoma.** Brit. J. Ophth. 41:377-382, June, 1957.

Prodromal malignant glaucoma may occur in the patient with shallow-angle glaucoma who has a small eye as measured by the size of the cornea or an abnormally large lens, as in acquired myopia. In extreme hyperopes or very old patients or with variations of tension in chronic narrow-angle glaucoma this abnormality should be suspected. Since the size of the lens is too great for the eye, the removal of the lens coupled with a filtering procedure such as iridencleisis is the procedure of choice. Where indicated, the lens extraction may be done first and the filtering procedure later. Six examples are described. (1 table, 29 references)

Lawrence L. Garner.

Dymshitz, L. **Pupillometric observations in glaucoma.** Oftal. Zhurnal. 1:21-24, 1957.

The author examined 25 hospitalized patients with glaucoma; 3 had simple glaucoma and 22 inflammatory glaucoma in various stages. In all the patients the size of the pupil was measured a number of times with Haab's pupillometer before the ocular tension was taken. The size of the pupil had no bearing on the lowering of the tension; the same fluctuation of the tension was observed in patients with a narrow pupil caused by miotics. There is no regular parallelism between the size of the pupil and the ocular tension in primary glaucoma. The enlargement of the pupil as a rule (with some exceptions) is not the cause of the increased tension, but a result of it. Wide fluctuation of tension in glaucoma may take place with fixed, often very narrow, pupils. The dilatation of the pupil in glaucoma is one of the factors which may endanger the eye, but it is a secondary phenomenon. The therapeutic effect of miotics is on the neurovascular mechanism of the eye, rather than in the production of miosis.

Olga Sitchevska.

François, J., Verriest, C. and de Rouck, A. **The visual functions in congenital glaucoma.** *Ann. d'ocul.* 190:81-107, Feb., 1957.

The basic premise of the authors is that while closed-angle glaucoma is a disease of the anterior segment, open-angle and congenital glaucoma are felt to be aberrations of the posterior segment, optic nerve and tract. They briefly list the functional disturbances in open-angle glaucoma and then report in detail studies of 22 cases of congenital glaucoma. All except one showed gonioscopic evidence of altered angle structure. The authors found frequent central scotomata which they believe due to macular aplasia. Nerve fiber defects were rarely seen, while general constriction of the field was common. Dark adaptation was affected, generally showing an elevation of the two segments of the curve. Two cases showed a dyschromatopsia. Of 13 patients examined electroretinographically the ERG was normal in only two. On the other hand electroencephalograms were routinely normal. The authors conclude that the functional defects of congenital glaucoma differ from those of open-angle glaucoma in that there is probably a lesion in the optic nerve in the latter disease. (7 figures, 33 references) David Shoch.

McBain, E. H. **Tonometer calibration.** *A.M.A. Arch. Ophth.* 57:520-531, April, 1957.

The author used normal eye-bank eyes for experiments to present new data on P_t (ocular pressure while tonometer is on the eye), measurements. Using the strain gauge method of Grant he was able to add further support to Friedenwald's contention that the simple formula $W/P_t = a + bR_t$ is accurate. His data indicated that measurements on animal eyes should not be included in calculation to calibrate tonometers for human eyes. Dis-

crepancies in the 1954 calibration scale can be obviated by using new coefficient values in the P_t formula obtained by this study. (5 tables, 3 references)

George S. Tyner.

Redslob, E. **The problem of optic nerve atrophy in chronic glaucoma.** *Ann. d'ocul.* 190:261-267, April, 1957.

It has been generally accepted that the optic atrophy of glaucoma results from a compression of the optic nerve head and the retinal ganglion cells as a result of increased intraocular pressure. Redslob takes issue with this hypothesis on several grounds. He states that glaucomatous optic atrophy has been frequently reported without elevations of tension and that in the usual case of glaucomatous atrophy the optic nerve is much more atrophic behind the cribriform plate than in front of it. He further quotes the work of Mantz who compressed the optic nerve of rats behind the globe and produced a "descending" optic atrophy. The author feels that the primary lesion of glaucomatous optic atrophy is an obliteration of the small vessels within the optic nerve resulting in a cephalo-ocular degeneration of the nerve rather than a degeneration in the reverse direction (oculo-cephalad) as has been generally assumed. (12 references) David Shoch.

Rougier, J., Chavanne, L., and Paufigue, L. **Malignant glaucoma after anti-glaucoma operations.** *Ann. d'ocul.* 190:268-280, April, 1957.

The authors review the history of malignant glaucoma and report that it usually occurs in eyes that are smaller than normal with relatively large lenses. In such eyes antiglaucoma operations are followed by flat anterior chambers and gradual swelling of the lens. This closes off all avenues of escape of aqueous humor and the tension gradually climbs

higher and higher and is resistant to all medical therapy. The authors support Chandler's view that the treatment of choice in such cases is an immediate lens extraction. They present four cases to support this view. (39 references)

David Shoch.

Skotnicki, Henryk. **Experimental glaucoma.** *Klinika Oczna* 27:27-36, 1957.

Unfortunately there is no disease in laboratory animals which would have the picture of human glaucoma. Attempts to produce it artificially either resulted in secondary glaucoma due to an inflammatory condition or were not stable enough to produce pathologic changes similar to those of glaucoma. The author feels that only steady pressure all around the globe in the equatorial region can produce glaucomatous changes. Rabbits were used for the experiment. A cotton ribbon 8 mm. wide was placed under the eye muscles over the equator and tightened with silk threads. Pressure rose to 65 to 70 mm. Hg. After a few days the cornea became hazy but gradually cleared. Within four months considerable changes could be seen ophthalmoscopically. Six months after application of pressure the eye was examined microscopically. It showed some cupping of the disc and atrophic changes in the nervous tissue but no inflammatory changes. The author feels that his method of increasing intraocular pressure may be valuable in studying problems of glaucoma. (4 figures, 3 references)

Sylvan Brandon.

Solarski, Zbigniew. **Glaucoma and flame nevus of the face.** *Klinika Oczna* 27:55-58, 1957.

A case of a flame nevus of the face associated with glaucoma of the same side is presented. In a man, 21 years of age, there was right-sided flame nevus in the area of the face supplied by the trigeminal

nerve. The right eye had absolute glaucoma and considerably distended conjunctival and episcleral vessels. Glaucoma had developed at the age of 15 years. The eye measurements were normal. The author feels that it was a case of Sturge-Weber syndrome without involvement of the nervous system. Glaucoma appeared at puberty because compensation in circulation of intraocular fluid failed at that time. Influence of sex hormones on the vasomotor center and on hyperplasia of blood vessels is considered probable. (7 references)

Sylvan Brandon.

Ustinova, E. **The permeability of blood vessels in glaucoma.** *Oftal. Zhurnal* 1:31-34, 1957.

The author examined the permeability of the capillaries on 100 glaucomatous and 100 control eyes. Amsler's fluorescein test was used for the eye blood vessels. The age and general condition in both groups were about the same; 73 patients had inflammatory and 27 had chronic simple glaucoma. In each group 62 to 65 patients had cardiovascular disease.

The permeability of the intraocular blood vessels was tested on 43 eyes of which 16 served as control; 13 eyes had glaucoma, in five eyes glaucoma was suspected and in five eyes there was iridocyclitis. Fluorescein appeared in the aqueous in 8 minutes after the intravenous injection, with maximal concentration in 20 to 22 minutes. There is no appreciable difference between the condition of the general permeability of the intraocular vessels in glaucomatous and nonglaucomatous eyes. There was no correlation between the form, stage, or rate of compensation of glaucoma and the general permeability of the blood capillaries. The increase of the permeability of the capillaries of 33 glaucomatous patients was probably due to cardiovascular dis-

ease. The permeability of the intraocular vessels is not increased in all glaucomatous eyes. (2 tables) Olga Sitchevska.

10

CRYSTALLINE LENS

Epstein, Edward. **The Ridley lens implant.** *Brit. J. Ophth.* 41:368-376, June, 1957.

The technical details of the Ridley lens implantation behind the iris are described with slight modification. The results of 84 such operations are discussed; 12 resulted in failure. Needling of an anterior occluding pupillary membrane was required in 15 cases and posterior needling was done in 24 cases. The use of a hydrocortisone suspension seems to reduce the intense postoperative iritis; the free alcohol compound is superior to the hydrocortisone acetate. It is dropped into the anterior chamber at the end of the operation and has made a definite difference in the reactions noted in this report. In many of the cases the lenses have dropped toward the 6-o'clock position. A modified lens is suggested for better refractive results; it facilitates centration and reduces some of the effects of posterior synechiae. (5 figures)

Lawrence L. Garner.

Owens, W. C. **The lens and the vitreous.** *A.M.A. Arch. Ophth.* 57:611-627, April, 1957.

This review presents the important developments in this field as reported in the literature in 1956. (147 references)

George S. Tyner.

Ridley, Frederick. **Safety requirements for acrylic implants.** *Brit. J. Ophth.* 41:359-367, June, 1957.

The necessary qualities of implant material and the conditions of manufacture are discussed. A method has been devised to keep the implants sterile in containers and the procedure of handling this

container at the operating table is outlined. The container is a sealed polyethylene tube containing a hard glass tube of sodium 0.5 percent bicarbonate in distilled water which has been autoclaved after sealing, and a pair of sterile (dry sterilized) forceps in a sealed glass tube. The main container is filled with 0.1 percent caustic soda. When the container is opened the solution is poured off and replaced by 0.5 percent sodium bicarbonate which eliminates any injury from the caustic soda solution. Since the acrylic lens may absorb injurious chemicals it is best for the surgeon to wear no gloves with powder, and to use no detergents. It is advisable to handle the implant with dry forceps. (4 figures, 5 references)

Lawrence L. Garner.

Ridley, Harold. **Anterior chamber lenticular implant.** *Brit. J. Ophth.* 41:355-358, June, 1957.

Because of the late dislocations of the implant and the prolonged stubborn iritis which sometimes follow the use of the intraocular implant first described, Ridley, describes an acrylic lens which is now implanted into the chamber anterior to the iris. The advantages of a lens in this position include the simpler technique and the fact that an intracapsular lens extraction can now be employed. Posterior dislocation of this lens is practically impossible, even with total iridectomy in the usual position. The lens can be inserted at the time of the extraction or at any later date. Less inflammation is noted when implantation is done later as a two-stage procedure. A special anterior chamber lens is ground for each case using the back vertex power and vertex distance of the aphakic correction. In monocular cases, the manufacturer requires the refraction of the normal eye. The present implant consists of a central lenticulus with three arms arranged for

support. When properly placed the lens lies midway between the cornea and iris. A special inserting forceps is required for insertion and when this operation is performed it is advisable to have several lenses of various size at hand. The vertical estimate of size is made by a caliper from the white of the upper limbus above to a similar point below. A haptic 0.5 mm. larger is then tried; if this is too difficult to insert, the next size 0.5 mm. smaller is used. An 8 mm. corneal incision is required. Insertion is made by placing the two lower legs into the lower part of the limbus and the single upper leg behind the corneoscleral lip. Reaction is minimal and controlled with corticosteroids. (3 figures, 7 references) Lawrence L. Garner.

Stokoe, N. L. **Soemmerring's ring**. Brit. J. Ophth. 41:348-354, June, 1957.

Soemmerring's ring, an after-cataract which results from peripheral proliferation of fibers, is discussed and reviewed. A case of spontaneous dislocation of this structure into the anterior chamber and its subsequent removal with excellent recovery are described. (6 figures, 15 references) Lawrence L. Garner.

11

RETINA AND VITREOUS

Bonavolonta, Aldo. **The macula in retinal detachment due to disinsertion**. Arch. di ottal. 61:5-18, Jan.-Feb., 1957.

The author reviews 66 cases of detachment from retinal disinsertion. Only 15.1 percent of the patients were myopes, in 60 percent the disinsertion was in the temporal sector (22.7 percent in the superior quadrant), in 24 percent trauma was recorded in the anamnesis, and in 18 percent the detachment was bilateral. In 42 percent of patients cystic degeneration of the macula was observed and in only 18 percent was successful operation followed by

improvement of the central vision. The severity of the macular degeneration depends on the length of time the detachment was present. (13 references)

John J. Stern.

Cramer, J. E. K., Bernasconi Cramer, E. R., Mercante, C. N., Iribarren, R. and Puppo, J. B. **Diagnosis and treatment of retinal detachment**. Arch. oftal. Buenos Aires 32:71-97, March, 1957.

Among the most valuable tools for the detection and precise location of tears are the Schepens binocular ophthalmoscope, especially when used in conjunction with Trantas' method of scleral depression, and the biomicroscopic examination of the fundus by means of both the Goldmann three-mirrored contact glass and the Hruby -55D lens. The study of the blood-aqueous barrier with the fluorescein permeability test may be of some assistance in that a marked increase in permeability is prognostically unfavorable. Tonographic readings were indicative of diminished outflow in the affected eyes; surgical cure, on the other hand, resulted in a return of the coefficient C to standard levels. (It might be well to note here that Grant and Trotter have found recently that in simple glaucoma the facility of outflow may improve to normal prior to retinal detachment.)

Techniques and indications of the diathermy, scleral resection, scleral buckling and vitreous implant operations are briefly described and discussed.

The histories of 52 diversely operated cases, of which 31 (59.6 percent) were cured, are presented in tabular form. (14 figures, 11 diagrams, 4 tables, 206 references) A. Urrets-Zavalía, Jr.

Gregory, Irene D. R. **Retinopathy of prematurity (retrolental fibroplasia)**. Brit. J. Ophth. 41:321-337, June, 1957.

Thirty children were examined and fol-

lowed in whom this condition had not progressed to total blindness. Myopic errors ranging from -2.5 to $-18D$ were found in 85 percent of the cases and nystagmus was found in 50 percent of the children with useful vision. Fourteen of the children had unilateral microphthalmos and the eye usually was amblyopic and deviated inward. Seventeen children (60 percent) had strabismus which, in all but one, was esotropia. Vitreous opacities were found in two thirds of the patients and malformations of the nervehead in one half. There were large areas of scarring and pigmentary changes in the fundus of most of the patients and small localized detachments of the retina were found in fourteen cases. Five of the eyes showed retinal folds. The high percentage of myopia found in this series of premature children strongly suggests a definite relationship between prematurity and myopia. (1 table, 25 references)

Lawrence L. Garner.

Horsten, G. P. M. and Winkelman, J. E. **Effect of temporary occlusion of the aorta on the electroretinogram.** A.M.A. Arch. Ophth. 57:557-565, April, 1957.

The authors found that the aorta of cats could be left clamped for about 10 minutes without producing retinal damage due to anoxia. (7 figures, 14 references)

George S. Tyner.

Kettesy, A. **Cure of a macular hole with light coagulation.** Klin. Monatsbl. f. Augenh. 130:465-471, 1957.

The author used the sun as light source. A 54-year-old woman with a macular hole was first exposed for one minute to bright sun at noon. The other eye was protected by filters but ensured fixation. Full correction was worn ($-14D$). This exposure was repeated while wearing a $-10D$ lens to cause a reaction on the detached retina itself. Finally, a third exposure was

done without any spectacles. The patient was then wheeled into the operating room and the subretinal fluid drained. A binocular mask was used for one week and a pinhole glass for another week. The result was excellent. (2 figures, 2 references)

Frederick C. Blodi.

Palich-Szanto, Olga. **Cystic, disseminated retinal edema in toxemia of pregnancy.** Klin. Monatsbl. f. Augenh. 130:523-528, 1957.

In both fundi numerous, greenish, round to oval foci were observed. They lay beneath the retinal vessels and seemed to be elevated. The arterioles were attenuated. The pregnancy had to be interrupted. (2 figures, 22 references)

Frederick C. Blodi.

Pau, Hans. **The histology of "cytoid degeneration" of the periphery of the retina.** Arch. f. Ophth. 158:558-567, 1957.

Pau's histologic studies show that the so-called cytoid areas (Blessig-Iwanoff edema) are in reality artifacts that are the result of histologic preparation and are not manifestations of retinal destruction which might have occurred intra vitam. Pau points out the differences between these changes and areas of vacuoles which occur as precursors of retinal detachment and are also found in the detached retina, in cyclitis and in other pathologic processes. (9 figures, 20 references)

F. H. Haessler.

Rendahl, I. **The electroretinogram in detachment of the retina.** A.M.A. Arch. Ophth. 57:566-576, April, 1957.

The author proves the contention that there is apparently an inherent predisposition to retinal separation in certain eyes. A poor E.R.G. is apparently associated with a poor prognosis. (3 figures, 6 tables, 16 references)

George S. Tyner.

Skeehan, R. A., Jr., Passmore, J. W. and Armington, J. C. **Retinitis pigmentosa with normal electroretinogram.** A.M.A. Arch. Ophth. 57:536-538, April, 1957.

On the basis of one case, the authors conclude that the E.R.G. cannot be relied upon as a completely conclusive proof of retinitis pigmentosa. (6 figures, 5 references)

George S. Tyner.

Tucker, D. P., Steinberg, A. G. and Cogan, D. G. **Frequency of genetic transmission of sporadic retinoblastoma.** A.M.A. Arch. Ophth. 57:532-535, April, 1957.

After tracing the pedigrees of 8 patients classified as "sporadic cases" of retinoblastoma, the authors conclude that these individuals have a one in four chance of transmitting the disease to 40 to 50 percent of their progeny. (2 figures, 8 references)

George S. Tyner.

Wolter, J. R. **Retinitis pigmentosa.** A.M.A. Arch. Ophth. 57:539-553, April, 1957.

The author employed the silver carbonate methods of del Rio Hortega to demonstrate new details of the pathology of retinitis pigmentosa. Both eyes of a 75-year-old man with advanced retinitis pigmentosa were studied. Many interesting features are reported along with a complete review of the literature. Among these interesting features are that the waxy appearance of the disc is due to the effect of a new-formed layer of astrocytes on the inner surface of the papilla and nerve fiber degeneration and gliosis within the nerve head. The nerve cells near the pigment layer of the retina were the most disturbed; almost all the visual cells had disappeared and a few cones were present only in the central area. Typical rosettes were found in the glial

scar of the outer layer of the central retina. (18 figures, 39 references)

George S. Tyner.

Zucolli, A. **Idiopathic cysts of the retina.** Bull. et Mém. Soc. franc. d'opht. 69:103-115, 1956.

Two varieties of so-called primary retinal cysts are described. The peripheral cysts of the ora serrata mostly are bilateral, symmetrical and located in the temporal inferior quadrant. They are round, sharply outlined and have a smooth, shiny surface. They are transparent and the bluish shimmer of the cyst itself contrasts sharply with the dark bands of the vessels on their surface. The cysts at the posterior pole are mostly monocular and present either an isolated bulla or a transparent globular mass, sharply delineated from the otherwise normal retina.

Two case histories are reviewed in detail. In a 46-year-old man both eyes were microphthalmic and highly hypermetropic. The retinal cysts were temporal and below, were clear and appeared greatly elevated. They were closely observed for six years and no tears, hemorrhages or pigmentation was seen. After this time a spontaneous partial collapse of one cyst was noted with improvement of the peripheral fields. The coexistence of a very high hypermetropia with this type of cyst pointed to a special disposition of the retina in abnormally small eyes, especially in a region normally not as well nourished as other parts and only vascularized during the last period of embryonic life. The cysts are probably of congenital, dysgenetic origin and begin in the intergranular layer. A pathologic report on similar cysts, published by J. François and Rabaey showed that these cysts originated from the integration of many small cysts. Their posterior wall consisted of the degenerated layer of rods and cones. The anterior wall consisted of glial tissue

and connective tissue fibers around several vessels. Toward the margin of the cyst strands of glial tissue connected the two walls. Pseudocysts, cystic tumors and intraocular parasites should be considered in the differential diagnosis. Strict observation for possible complications, like disinsertions and perforations of the external wall due to necrosis is always indicated. In a quiet idiopathic cyst surgery should not be performed. (5 figures, 14 references) Alice R. Deutsch.

12

OPTIC NERVE AND CHIASM

Bregeat, Paul. **Papilledema. Report read at the meeting of the Soc. franç. d'opht., May 8th, 1956.** Bull. et Mém. Soc. franç. d'opht. 69:1-22, 1956.

The difficulties in the differential diagnosis of concomitant papilledema, primary or essential papilledema, congenital anomalies of the vessels, the nervous and mesodermal tissues of the disc and high refractive anomalies are outlined. It is emphasized that this differential diagnosis is essential for the evaluation of the physiopathology of the clinical picture, for the prognosis and for the course of treatment. The ophthalmoscopic pictures of papilledema, papillitis and the accompanying functional disturbances are described. A revision of the classical signs and symptoms of papilledema was found to be indicated. Normal visual acuity, eccentric defects in the visual fields in form of cuneiform, arciform and quadrantic scotomas are referable to papillitis as well as to papilledema, while an enlargement of the blind spot, sloping border and disorders in spatial summation denote papilledema. A swelling of the disc over 3D can occasionally be seen also in papillitis. The detachment of the prepapillary limitans interna without precipitates and without Tyndall phenomenon is charac-

teristic for papilledema. The possible causes of papilledema, more or less typical for specific age groups, are reviewed together with the indication and interpretation of additional studies like E.E.G., E.R.G., ventriculography and angiography. It is the responsibility of the ophthalmologist to decide on the indications for and the feasibility of adequate medical or surgical treatment in cooperation with the neurologist. Good teamwork is essential to achieve a correct diagnosis and immediate specific treatment.

Alice R. Deutsch.

Dejean, C. **Atypical papilledema.** Bull. et Mém. Soc. franç. d'opht. 69:23-35, 1956.

Sixteen cases of "atypical papilledema" are analyzed. The first eight patients had a specific chorioretinitis. The disc showed a serous, not specific infiltration. The visual acuity remained more or less normal as long as the edema did not affect the posterior pole. The similarity to "choked disc" was striking. The presence of an intra-cranial lesion was excluded. Active antituberculosis treatment was effective and confirmed the diagnosis. Similar serous infiltrations of the disc were seen after trauma and sometimes persisted for a long time. Papilledema was also observed simultaneously with an optico-chiasmatic arachnoiditis and not only with the cystic form with its potential accompanying intracranial hypertension but also with the simple cicatricial variety and its fibrous strands around the optic nerve. Papilledema also occurred in two hypertensive patients and no other causes were found to explain the papilledema. Another patient had a bilateral papilledema during a period of eight years without any functional loss and was in good general health.

There are many theories on the development of papilledema; however, they only fit certain categories. The details on

which these are based are summarized.
(10 figures)

Alice R. Deutsch.

François, J., Verriest, G. and Baron, A.
Vascular pseudopapillitis. Bull. et Mém.
Soc. franç. d'opht. 69:36-57, 1956.

Pseudopapillitis of vascular origin is a disease of the optic nerve, caused by a sudden occlusion of one or more of the nutritive vessels of either one or both segments of the optic nerve, namely the intraocular and the juxtabulbar segments. The signs and symptoms resemble those of classical papillitis and include papilledema, impairment of central vision and characteristic defects in the visual fields. Etiologic examinations are often negative except for the presence of vascular anomalies. The course of the disease follows a typical clinical pattern. Occasional prodromal disturbances occur, but mostly the more or less complete loss of vision is rather sudden. The disc shows a partial or total edema with or without affection of the peripapillary area. The arteries are narrow and sclerosed, the veins more or less dilated. There are no other abnormalities in the retina. In the presence of a cilioretinal artery the corresponding retinal area shows the signs characteristic of an arterial embolism. The ensuing atrophy is mostly total, with sharp margins, narrow arteries and sheathing of the arteries, on the disc and in the peripapillary region. The extensive and concentrated studies of the authors concerning the blood supply of the optic nerve and its independence from the retinal artery itself are fundamental to an understanding of these and allied pathologic changes. The sudden loss of vision might be referred to an occlusion of the central artery of the optic nerve. A part of the peripheral field might be preserved because of the blood-supply through the pial vessels and Zinn-Haller-circle with anas-

tomoses. The pseudopapillitis of vascular origin was observed in the presence of arteriosclerosis, temporal arteritis and exceptionally in polyarteritis nodosa, Reynaud's disease and polycythemia vera.

Seven case histories are reviewed in detail. Six patients had disseminated arteriosclerosis with abnormal EEG, ERG and ECG. The seventh patient had an arteritis of the left temporal artery. First the right and a few days later the left central optic artery became occluded, with resulting blindness. The prognosis for the preservation of vision in vascular disease of the optic nerve is generally poor. Neither medical nor surgical treatment has been effective. (9 figures, 40 references)

Alice R. Deutsch.

Jayle, G.-E., Aubert, L. and Boyer, R.
Adaptometric, campimetric, and electrophysiologic investigation of two cases of retrobulbar optic neuritis. Ann. d'ocul. 190:117-129, Feb., 1957.

The authors continue their investigation of visual field testing in moderate illumination (mesopic campimetry). They report two cases of retrobulbar neuritis. In both cases photopic campimetry was normal and scotopic campimetry not precise. Dark adaptation curves were normal in both cases but there were definite deficiencies in retinocortical conductivity. (6 figures)

David Shoch.

Wertheimer, J. and Wertheimer, D.
Sequelae of papilledema in cerebral tumors. Bull. et Mém. Soc. franç. d'opht. 69:67-73, 1956.

A statistical review is presented of 90 cases of brain tumor as observed and operated on at the Neuro-surgical Center at Lyon. Every patient had either bilateral or monocular papilledema. The pathologic diagnosis could be made in most cases. Astrocytomas and meningiomas gave a better prognosis than other tumors. The

degree of elevation of the disc was found to have no localizing value. The presence of papilledema always should hasten additional related investigations; a delay of necessary surgery for more than three months invariably was followed by discoloration of the discs and more or less impairment of ocular function. (1 table)

Alice R. Deutsch.

13

NEURO-OPHTHALMOLOGY

Danis, P. and van Eyck, M. **Intracranial hypertension and transitory external oculomotor paralysis following otitis media (otic hydrocephalus, meningeal hydrops).** Bull. et Mém. Soc. franç. d'opht. 69:58-66, 1956.

Meningeal hydrops following disease of the middle ear and mastoid has only been rarely discussed in ophthalmologic literature, in spite of the repeated observation of associated ocular signs and symptoms. The author reports a corresponding case because it provided noteworthy diagnostic and therapeutic problems. A nine-year-old child developed bilateral papilledema and a bilateral abducens paralysis one month after a radical mastoidectomy and intensive antibiotic treatment. Local otologic and general medical examinations were negative. The child had no headaches and had no subjective complaints except diplopia. The papilledema increased for one week and later slowly receded spontaneously until the discs were normal two months later. The diplopia only improved after repeated lumbar punctures. The spinal fluid was normal in composition but under increased pressure (900 mm. water) at the time of the first lumbar puncture. The diagnosis of otitic hydrocephalus was confirmed after reviewing Gradenigo's syndrome, otitic brain abscess and septic meningitis and encephalitis in the differ-

ential diagnosis. The prognosis of otitic hydrocephalus is good. Complete investigations with their inherent risks mostly are not warranted. (1 figure, 52 references)

Alice R. Deutsch.

Passow, A. **Neurogenic diseases, symptoms and cause and effect on the focus.** Klin. Monatsbl. f. Augenh. 130:433-464, 1957.

This is a review of some of the author's earlier papers. Heterochromia may be associated with a status dysraphicus. The sympathetic heterochromia can experimentally be produced in young cats. The author speculates on the neurovegetative cause of congenital lens luxation, corectopia, fifth-nerve palsy, and neuroparalytic keratopathy. (1 table, 64 references)

Frederick C. Blodi.

Pigasson, R., Garipny, M. M. and Lazorthes. **Statistical reports of the Neurological Center at Toulouse. Intracranial hypertension, ophthalmoscopy and venous pressure.** Bull et Mém Soc. franç. d'opht. 69:74-76, 1956.

The survey presented includes 350 patients of whom 263 showed no venous pulsation. In these patients pulsation of the veins could not be provoked. Intracranial hypertension was confirmed by ventriculography, arteriography or surgery. 97 patients had spontaneous venous pulsation. Additional investigations substantiated the absence of intracranial hypertension. Several of the patients who were seen early developed a papilledema soon after the lack of venous pulsation was noted. Absence of pulsation of the central veins should be considered significant for the diagnosis of intracranial hypertension. At the beginning of the investigations the pressure on the eyeballs was made with Bailliart's dynamometer; later on only digital pressure was used. (2 tables)

Alice R. Deutsch.

14

EYEBALL, ORBIT, SINUSES

Linnen, Hans Joseph. **A modification of the Kroenlein operation and the surgical treatment of malignant exophthalmos.** Klin. Monatsbl. f. Augenh. 130:471-483, 1957.

This operation is advised for desperate cases and was done on two patients who had not benefited from a previous trans-frontal decompression. In this operation the greater part of the lateral orbital wall is resected. (10 figures, 21 references)

Frederick C. Blodi.

Manzitti, E. **Report of a case of unilateral exophthalmos due to primitive ethmoidal tumor.** Arch. oftal. Buenos Aires 32:59-61, Feb., 1957.

A nine-year-old girl had a rapidly growing left proptosis for the last few months. Marked exophthalmos, papilledema and restricted adduction of the affected eye were found. Digital pressure exerted upon the globe was painless but met a hard resistance in the depths of the orbit, in which, however, no definite mass could be palpated directly. Vision in the left eye was reduced to light perception. The left frontal, ethmoidal and sphenoidal sinuses were radiographically opaque and the inner orbital wall showed some destruction. Surgical exploration was performed through a Killian approach; a tumor process was disclosed and a biopsy was taken. Pathologically, a diagnosis of undifferentiated sarcoma was made and the patient submitted to roentgenotherapy. (3 figures)

A. Urrets-Zavalía, Jr.

McCoy, Frederick J. **Management of the orbit in facial fractures.** Plastic & Reconstructive Surgery 19:236-245, March, 1957.

For lateral fractures, the author uses

the Gillies approach with a number 21 cervical sound beneath the temporal fascia and exerts upward pressure against the depressed fragment until a snap is felt. If additional stabilization is necessary a Kirschner wire is used.

For fractures of the infraorbital rim he reestablishes the floor by open reduction with direct wiring of the fragments, especially if extensive comminution is present. He uses a transantral exposure if there are dislocated fragments in the orbital floor and packs the antrum to help stabilize the fragments.

For medial fractures he makes two angular incisions just anterior to the inner canthi and retracts the lacrimal apparatus and subadjacent soft parts to admit the rubber-shod blade of the Asch forceps on either side. He reduces the fracture forcibly, using considerable force and closes up the naso-ethmoid arch.

When treatment has been delayed for three weeks or more, he uses the incisions described to gain access to the posterior border of the frontal process of the maxilla. Drill holes are made on either side through which the wire is passed on a large curved needle. He exerts pressure on the wire to produce a medial pull on the fragments and head cap traction is applied. He continues the traction from four to six weeks.

When reduction has been delayed for more than six weeks he refractures the improperly healed fragments with a chisel and strips the orbital septum free from the periorbital attachment for 2 cms. above and below to obtain relaxation. (10 figures, 9 references)

Alston Calahan.

15

EYELIDS, LACRIMAL APPARATUS

Couzi, Jaques. **Relation between pathologic Meibomian gland hypersecretion**

and intolerance to fluorescent lighting. *Ann. d'ocul.* 190:130-134, Feb., 1957.

Complaints of intolerance to fluorescent lighting are very common and in almost all cases this is associated with a Meibomian gland hypersecretion. Most of these people get relief from wearing tinted lenses of a blue-green shade. It has been suggested that these lenses filter out the infrared rays, but the author points out that fluorescent light contains less infrared than ordinary incandescent light. He feels that the problem is simply one of dazzling and that the tinted lenses cut down the dazzle. (1 table, 9 references)

David Shoch.

Drozdowska, Stanislaw. **Surgical treatment of entropion.** *Klinika Oczna* 27:59-61, 1957.

Correction of entropion is attempted by transplanting a narrow strip of skin removed from the upper lid into a groove made in the lid margin. The gray line is split to the depth of 2 or 3 mm. and the graft is sutured into it. The author was successful in 20 cases; in one patient with heavy trachoma scars entropion remained uncorrected. (5 figures)

Sylvan Brandon.

Nover, Arno. **Ectopic lacrimal glands.** *Klin. Monatsbl. f. Augenh.* 130:483-488, 1957.

Four cases of epibulbar tumor are reported which proved on histologic examination to consist of lacrimal gland tissue. The occurrence of this tissue is explained by the migration of these glands from the lower lid upward during embryonic development. Twenty caruncles were obtained at autopsy and in eight of them serous glandular elements could be found. (4 figures, 18 references).

Frederick C. Blodi.

Szmyt, Jacek. **Radiography of the nor-**

mal lacrimal passages. *Klinika Oczna* 27:37-42, 1957.

To obtain good radiograms of the lacrimal pathways the picture should be taken immediately after injection of the contrasting medium. Canaliculi then are not usually visible. Lacrimal sacs may be small (15 sq. mm.), medium (29 sq. mm.) and large (45 sq. mm.). They may be oval and round and may be located vertically or obliquely. The lacrimal sac may be well outlined or it may join the tear duct without a definite step. Tear ducts normally may be wide or narrow but there is no difference between the parts in the bone and in the nasal mucous membrane. The size and the shape of the opening into the nose affect the appearance of the contrast medium in the nose. X-ray study of the lacrimal passages has considerable diagnostic and prognostic value. (11 figures, 2 references)

Sylvan Brandon.

16

TUMORS

Dunphy, E. B., Dowling, J. L. Jr., and Scott, A. **Experiences with radioactive phosphorus in tumor detection.** *A.M.A. Arch. Ophth.* 57:485-490, April, 1957.

The authors report their experience with the P^{32} test in a series of 50 cases of suspected or proved intraocular malignancy. Tumors of the choroid and ciliary body which were accessible to the Geiger counter and gave high counts proved to be malignant by pathological examination. In six proved cases, where the tumor was located far posteriorly, the P^{32} test was negative.

In four out of five proved malignant melanomas of the iris the test was positive. In five eyes with benign lesions the test was negative. In retinoblastoma the test was inconclusive. Several pitfalls in the technique of using the test are outlined. (3 figures, 10 references)

George S. Tyner.

Gärtner, J. **Retinoblastoma and medulloblastoma. A comparison of their morphologic and biologic characteristics.** Arch. f. Ophth. 158:605-617, 1957.

These tumors are held to be identical histologically and biologically but it emerges that the occurrence of "true rosettes" is not a valid differential characteristic because the appearance of these structures depends on the age and the grade of differentiation of the tumor. The author's extensive study makes it clear that the tumor of the pars optica retinae can no longer be looked upon as a medulloblastoma which is localized in the retina. It is in reality a unique type of tumor. (3 figures, 33 references)

F. H. Haessler.

Kurtzman, J. L. **Embryotoxon with associated hemangioma of the globe.** A.M.A. Arch. Ophth. 57:590-592, April, 1957.

A case in which these two abnormalities coexisted is reported. (2 figures, 8 references)

George S. Tyner.

Sbordone, G. **A case of angioma of the orbit.** Arch. di ottal. 60:338-343, Nov.-Dec., 1956.

A case of angioma of the orbit without subjective or ophthalmoscopic changes is reported. (3 figures, 11 references)

John J. Stern.

Valvo, Giuseppe. **Epithelioma of the**

limbus. Arch. di ottal. 60:322-337, Nov.-Dec., 1956.

A case of epithelioma at the limbus is reported. (8 figures, 12 references)

John J. Stern.

17

INJURIES

Alajmo, Arnaldo. **Some clinical aspects of ocular trauma in boxers.** Arch. di ottal. 61:73-80, Jan.-Feb., 1957.

The literature is reviewed and the problem is discussed without introducing any new data or concepts. (6 references)

John J. Stern.

Arouh, Julio. **Bilateral keratectasia due to self-inflicted trauma.** Arch. oftal. Buenos Aires 32:5-8, Jan., 1957.

In a 33-year-old unmarried woman, who shortly after puberty acquired an irresistible habit of compressing violently her eyeballs with her thumbs, a bilateral central protrusion of the cornea with opacification was present. Vision was reduced to 0.2 in both eyes. This case of self-abuse through physical excitation represented apparently a sexual deviation in a Freudian sense where, owing to a migration of the erogenous areas, the manipulation of normally inert parts led to some kind of orgasm. The patient had to be finally secluded in an institution for the treatment of mental disorders. (1 figure)

A. Urrets-Zavalía, Jr.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

DEATHS

John Walker Fairing, Shaker Heights, Ohio, died April 12, 1957, aged 84 years.

Halvor Larson Harley, Atlantic City, New Jersey, died March 16, 1957, aged 74 years.

Mark Walton Jacoby, Cleveland, Ohio, died March 28, 1957, aged 64 years.

Francis Xavier Siegel, Cincinnati, Ohio, died April 10, aged 75 years.

Frank Robert Spencer, Boulder, Colorado, died April 20, 1957, aged 77 years.

William Francis C. Steinbugler, Brooklyn, New York, died April 7, 1957, aged 70 years.

ANNOUNCEMENTS

SOUTHERN SECTION DINNER

On Tuesday, November 12, at 6:30 P.M., at the Surfcomber Hotel, Miami Beach, Florida, will be held a joint dinner meeting of the Southern Section of the Association for Research in Ophthalmology and the Eye, Ear, Nose, and Throat Section of the Southern Medical Association.

COURSE IN OPHTHALMIC SURGERY

An intensive postgraduate course in ophthalmic surgery will be given by the staff of the New York Eye and Ear Infirmary November 18 through 25, 1957. The course will cover retinal detachment, cataract, plastic, motility, and glaucoma surgery. There will be didactic lectures as well as demonstrations in the operating room. The fee will be \$250.00. For further details write:

Mrs. Mabel Stewart
218 Second Avenue
New York 3, New York

CENTRAL ILLINOIS MEETING

The 29th annual meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held in Springfield, Illinois, on September 27th, 28th, and 29th. Speakers for the meeting will be Dr. K. M. Simonton, Mayo Clinic, Rochester, Minnesota; Dr. Charles E. Iliff, Jr., Wilmer Institute, The Johns Hopkins Hospital, Baltimore; Dr. William Hubble and Dr. R. E. Knight, Bloomington, Illinois. Dr. S. Glidden Baldwin will be the Friday evening speaker and his subject will be "Four on safari."

GILL HOSPITAL

The Gill Memorial Eye, Ear, and Throat Hospital, Roanoke, Virginia, will hold its 31st annual spring congress in ophthalmology and otolaryn-

gology and allied specialties on April 14 through April 19, 1958.

Among the guest speakers invited to participate are: Dr. David B. Allman, Atlantic City, New Jersey; Dr. Edwin N. Broyles, Baltimore; Dr. John F. Conley, New York; Dr. George Crile, Jr., Cleveland; Dr. Fred W. Dixon, Cleveland; Prof. Dr. Gerhard Demagk, Germany; Dr. Leon Goldman, Cincinnati; Dr. Roscoe J. Kennedy, Cleveland; Dr. Perrin H. Long, Brooklyn; Dr. S. C. Missal, Cleveland; Dr. C. Stewart Nash, Rochester, New York; Dr. Edward W. D. Norton, New York; Dr. Donald M. Shafer, New York; Dr. Benjamin H. Shuster, Philadelphia; Dr. Byron Smith, New York; Dr. Richard C. Troutman, Brooklyn; Dr. Henry Wagener, Mayo Clinic, Rochester, Minnesota; Dr. Frank Walsh, Baltimore; Dr. James W. Watts, Washington, D.C.; Dr. Lorenz Zimmerman, Washington, D.C.; Dr. J. V. V. Nicholls, Montreal.

WILLS CLINICAL CONFERENCE

The 10th annual clinical conference of the Wills Eye Hospital staff and society of ex-presidents will be held in Philadelphia on Thursday, Friday, and Saturday, February 20, 21, and 22, 1958, in connection with the 125th anniversary of the founding of the hospital. In commemoration of the anniversary a number of attractive features are being planned by the committee for the early part of the week.

The Arthur J. Bedell lecture will be delivered by Dr. Paul Chandler on Saturday, February 22, 1958. In addition to individual presentations and panel discussions on clinical, surgical, and research subjects, the scientific program will include special surgical clinics.

The social activities will include certain features for the ladies as well as an informal reception and supper for all those who attend on Friday evening, February 21, 1958. The conference will be concluded with a dinner meeting of the society of ex-residents of the hospital.

OHIO POSTGRADUATE COURSE

A postgraduate course in ophthalmology will be given on March 3 and 4, 1958, in the Conference Theatre, Ohio Union Building, by the Department of Ophthalmology, Ohio State University, Columbus, Ohio.

Speakers for this course will include Dr. F. Bruce Fraclik, University of Michigan; Dr. Algon B. Reese, New York; Dr. Charles L. Schepens, Boston; and Dr. William H. Havener, Dr. Madge

T. Macklin, Dr. Torrence A. Makley, and Dr. William H. Saunders, Ohio State University.

The registration fee is \$20.00 and further details may be obtained from:

Dr. William H. Havener
Department of Ophthalmology
University Hospital
Columbus 10, Ohio

SOCIETIES

NEW YORK OFFICERS

The officers of the New York Society for Clinical Ophthalmology for the 1957-1958 season are:

President, Dr. Harvey E. Thorpe; vice-president, Dr. Arthur Linksz; recording secretary, Dr. Jesse M. Levitt; corresponding secretary, Dr. Leon H. Erlich; treasurer, Dr. Henry M. Kera; historian, Dr. Robert S. Coles.

Committee chairmen are: Program, Dr. Abraham Schlossman; instruction session, Dr. Alfred Kestenbaum; legislative, Dr. Benjamin C. Rosenthal; membership, Dr. Howard Agatston; industrial, Dr. Edward M. Douglas.

Dr. Max Chamlin, the retiring president, was elected to the advisory council.

AMERICAN OPHTHALMOLOGICAL SOCIETY

At its annual meeting, the American Ophthalmological Society elected the following officers for the coming year: President, Dr. Walter S. Atkinson, Watertown, New York; vice-president, Dr. Derrick Vail, Chicago; secretary-treasurer, Dr. Maynard C. Wheeler, New York; editor of the *Transactions*, Dr. Gordon M. Bruce, New York.

PERSONALS

Dr. Hedwig S. Kuhn, Hammond, Indiana, presented a paper on industrial ophthalmology at the XII International Congress of Industrial Medicine in Helsinki, Finland, on July 2nd. Dr. Kuhn will also be a guest speaker at the meeting of the Australian Ophthalmological Society in October.

Dr. S. Rodman Irvine, Beverly Hills, California, has been elected a trustee of Pomona College at Claremont, California.

Dr. Algernon B. Reese, New York, has been awarded the degree of Doctor of Laws, by Duke University, Durham, North Carolina.

E. B. Meyrowitz

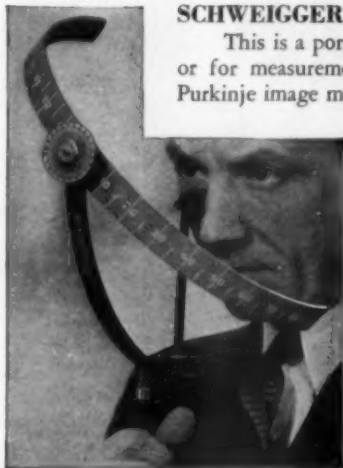
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NEW ORLEANS ACADEMY OF OPHTHALMOLOGY

The Eighth Annual meeting of the New Orleans Academy of Ophthalmology will be held in New Orleans in the Roosevelt Hotel—February 24-28, 1958, featuring "Symposium on Uveitis." The registration fee of \$75.00 includes associate membership in the Academy for the year 1958, as well as all other features of the convention. Hotel reservations should be made early by writing directly to the Executive Secretary, P.O. Box 469, New Orleans, La.

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